ANALYSIS OF CADAVER RENAL TRANSPLANT IN GOVERNMENT STANLEY HOSPITAL

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CERTIFICATE

This is to certify that the dissertation titled **"ANALYSIS OF CADAVER RENAL TRANSPLANT IN GOVERNMENT STANLEY HOSPITAL"** is the bonafide original work of **Dr. S. KRISHNA KUMAR,** in partial fulfillment of the requirements for D.M. Branch – III (Nephrology) Examination of the Tamilnadu DR. M.G.R Medical University to be held in AUGUST 2010. The Period of study was from October 2008 to April 2010.

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DECLARATION

I, **Dr. S. KRISHNA KUMAR,** solemnly declare that dissertation titled **"Analysis of Cadaver Renal Transplant in Government Stanley Hospital"** is a bonafide work done by me at Government Stanley Medical College and Hospital during October 2008 to April 2010 under the guidance and supervision of my unit chief **Prof. R.Vijayakumar, M.D., D.M.(Nephrology),** Professor and Head, Department of Nephrology, Government Stanley Medical College and Hospital, Chennai.

This dissertation is submitted to Tamilnadu Dr. M.G.R Medical University, towards partial fulfillment of requirement for the award of **D.M. Degree (Branch – III) in Nephrology – August 2010.**

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CONTENTS

INTRODUCTION

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Treatment option for Chronic Kidney Disease-Stage5 (CKD-stage5) patients fall into three categories viz., Haemodialysis, Peritoneal dialysis and Renal Transplantation. Many studies proved that the kidney transplantation is distinctly superior and it is associated with reduced mortality and morbidity compared to haemodialysis or peritoneal dialysis[3 – 16].

The renal donors are of three types viz. live related, live unrelated and cadaver. With nuclear families, working members in the family and the increased prevalence of diabetes mellitus and hypertension among general population, it is difficult for the CKD-stage5 patients to get suitable willing live donors. The only option for them will be cadaver donors.

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AIM OF THE STUDY

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- To evaluate the short term outcome of recipients of deceased donor grafts.

REVIEW OF LITERATURE

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The most clinically useful method of assessing renal transplantation outcomes is measurement of allograft survival. Other important measures include allograft function (typically measured by serum creatinine), patient survival, number and severity of acute rejection episodes, days of hospitalization, and quality of life indices. Most of the data for assessing transplant outcome is from United States Renal Database System (USRDS)[1], Collaborative Transplant Study (CTS)[2] and Australia and New Zealand Dialysis and Transplant (ANZDATA) Registry.

Actual and Actuarial Allograft and Patient Survival

Allograft survival is calculated from the day of transplantation to the day of reaching a defined endpoint (i.e., return to dialysis, retransplantation, or death, whichever occurs first). In practice, survival is usually calculated by actuarial methods. These methods imply estimation or projection of survival since not all patients will have been followed for the same period of time. Also, since not all patients will have reached the defined endpoint, censoring of such patients is required. Projected survival estimates must be interpreted with caution; projected survival may ultimately not be as impressive as actual survival[11]. Another actuarial measure commonly used is graft half life. Graft half life is the number of years before 50% of the graft, that survive at one year will fail or the patient will die with a functioning graft.

Traditionally, graft survival is assessed under two distinct time phases: early and late. Early graft loss refers to loss in the first 12 months and late loss to any time thereafter. In the first 12 months, graft loss is not rare because of technical complications such as graft thrombosis and severe rejection. After 12 months, the incidence of graft loss is lower but remains quite stable over time. Usually, analysis of long term survival is restricted to those allografts that have survived to 12 months post-transplantation. By this definition, patient death is equivalent to graft loss. Graft survival can also be calculated after censoring for patient death. Death with a functioning allograft is not necessarily a bad outcome and in fact is probably the best outcome, provided survival after transplantation is prolonged.

SHORT TERM OUTCOMES IN RENAL TRANSPLANTATION

The principal causes of graft loss in the first post-transplantation year are acute rejection, graft vessel thrombosis, primary nonfunction, sepsis and patient death. The current adjusted one year survival probability for recipients of deceased donor allografts (first or subsequent transplant) is 91%; this has slowly but steadily improved over the past 25 years. The principal causes of patient death in the first year are cardiovascular disease and infection.

LONG TERM OUTCOMES IN RENAL TRANSPLANTATION

There has also been a steady improvement in long term allograft survival. Recently this increase has occurred mainly in higher risk patients, such as those undergoing retransplantation. Beyond the first post-transplantation year, the principal causes of renal allograft loss are patient death and chronic allograft

nephropathy; less common causes are late acute rejection and recurrent disease[8]. Chronic allograft nephropathy is a nonspecific term and in practice often encompasses chronic damage due to ischemia, rejection and calcineurin inhibitor toxicity. The number one cause of death post-transplantation remains cardiovascular disease, followed by infection and malignancy. In children, however, death is a much less common cause of graft loss; conversely, in the elderly, it is more common.

FACTORS AFFECTING RENAL ALLOGRAFT SURVIVAL

Prospective studies and analyses of registry data have shown that many factors are associated with renal allograft survival. These can be considered as either donor, recipient, or donor-recipient.

Donor-Recipient Factors

Delayed Graft Function

Delayed graft function (DGF) is usually defined as failure of the renal allograft to function immediately post-transplantation, with the need for one or more dialysis sessions within a specified period, usually one week. DGF is associated with poorer graft survival, poorer graft function, and higher risk of patient death[10], in part because of the association of DGF with higher rates of acute rejection. Rejection may be more common because ischemia-reperfusion injury increases the immunogenicity of the graft. Most studies have also demonstrated that, even in the absence of documented acute rejection, DGF is associated with poorer long term graft function and survival[11].

Risk factors for DGF are:

- Donor age (>40 years)
- Cold ischemia time (>12 hrs)
- Recipient race
- PRA (>50%)
- HLA mismatch
- Duration of dialysis

HLA Matching

Registry data from many countries clearly demonstrate that, even with current immunosuppression regimens, better HLA matched deceased donor allografts still have better survival[18]. This is why many countries operate national or international sharing systems for zero-mismatched renal allografts, even though this prolongs cold ischemia times. The hazard ratio of graft failure for recipients of a zero-mismatched allograft in the 1998 to 2003 cohort was 0.85 (0.78 to 0.92) compared to 1.24 (1.15 to 1.34) for recipients of a three mismatched allograft[1]. The better outcomes are presumably related to fewer immunologic failures. There is some evidence, however, that the benefits of HLA matching are diminishing, probably because of more effective immunosuppression[18].

Cytomegalovirus Status of Donor and Recipient

Registry data show a small but definite effect of donor and recipient cytomegalovirus (CMV) serologic status on renal allograft and recipient survival[1].

Donor negative-recipient negative-pairing have the best outcomes, whereas donor positive-recipient negative pairing have the worst. CMV probably affects graft outcomes via overt infection, but subclinical effects on immune function may also be important.

Center Effect

Not surprisingly, outcomes have varied widely among transplantation centers. This reflects normal statistical variance as well as center expertise. It is important to note that outcomes will be confounded by many donor and recipient factors that differ across centers. Thus, between center comparisons are difficult. USRDS data suggest minimal difference in outcomes between small and large transplantation centers in the United States[45].

Donor Factors

The quality of the kidney immediately prior to transplantation has a major impact on long term graft function and the risk of developing chronic allograft nephropathy.

Donor Source : Deceased versus Living Donor

The donor source is one of the most important predictors of short and long term graft outcomes. In general, living donor grafts are superior to deceased donor grafts. The better healthy living donors, the absence of brain death, the general benefits of elective as opposed to semi emergency surgery, avoidance of ischemia-reperfusion injury, high nephron mass and probably the effects of a

shorter waiting time. Better compliance by the recipient in view of the relationship e.g., spouse, a care giver may also play a role.

Donor Age

Deceased donor and living donor allografts from those aged older than 50 years, and particularly older than 65 years, have poorer outcomes[1]. These results are thought to reflect a higher incidence of DGF and of "nephron underdosing". Grafts from older donors have fewer functioning nephrons because of the aging process and donor-related conditions such as hypertension and atherosclerosis.

Cold Ischemia Time

Prolonged cold ischemia time is associated with higher risk of DGF and poorer allograft survival[19]. Registry data suggest that >24 hours is particularly deleterious to the graft[1].

Donor Race

The survival of deceased donor grafts obtained from African-Americans is poorer than grafts from Caucasians. One theory is that a lower nephron number in African Americans is important.

Donor Sex

There is evidence that grafts from deceased females donors have slightly poorer survival, particularly in male recipients[1,20]. This probably reflects "nephron underdosing", as females have smaller renal mass than males. However, differences in the antigenicity of female grafts may also be a factor[19].

Donor Nephron Mass

An imbalance between the metabolic/excretory demands of the recipient and the functional transplant mass has been postulated to play a causative role in the development and progression of chronic allograft nephropathy. "Nephron underdosing", exacerbated by perioperative ischemic damage and postoperative nephrotoxic drugs, might lead to nephron overwork and eventual failure, similar to the mechanisms occurring in native kidney disease.

Expanded Criteria Donors

As the discrepancy between the number of patients awaiting kidney transplantation and the number of available organs increases, many countries are now using expanded criteria donor (ECD) allografts[21] previously named marginal kidney donor. ECD kidney is defined as a kidney from a deceased donor older than 60 years or aged 50 – 59 years with two additional risk factors including a history of hypertension, death due to CVA or elevated creatinine (>1.5 mg/dl). Survival of ECD kidneys is, on average, shorter than regular deceased donor kidneys for two general reasons : first, the baseline GFR of these kidneys is likely to be lower and, second, ECD kidneys tend to be transplanted into older recipients who have higher rates of post-transplantation death. However, it should be emphasized that transplantation with an ECD kidney always confers a significant survival advantage compared to remaining on the transplant waiting list (on dialysis for long)[10].

Other nontraditional donors are non-heart beating donors. The use of non-heart beating donors has been controversial as short term outcomes are inferior to those seen with standard deceased donor kidneys. This reflects the longer period of warm ischemia. Rates of DGF and primary nonfunction are generally higher than with standard donors.

Recipient Factors

Recipient Age

In general, graft survival rates are poorer in those at the extremes of age: younger than 17 and older than 65 years[1]. In the young, technical causes of graft loss such as vessel thrombosis are relatively more common. Acute rejection is also a more common cause of graft loss; conversely, death with a functioning graft is relatively rare.

The elderly (those older than 65 years) are forming an increasing percentage of the incident and prevalent Chronic Kidney Disease-stage5 population. Many of these patients have significant comorbid disease, particularly cardiovascular disease and type 2 diabetes mellitus. Nevertheless, age per se is not a contraindication to transplantation: among elderly patients carefully screened and deemed fit for the procedure, long term outcomes are clearly better with transplantation than dialysis[3]. It is, therefore, appropriate that transplantation in elderly recipients is becoming more common compared with younger recipients, death with a functioning graft is a more common cause of graft loss in the elderly (responsible for >50% of graft failures). Conversely, acute rejection may be less common. Thus, although randomized, controlled trials are not

available, it seems reasonable, in general, to use less aggressive immunosuppression in the elderly.

Recipient Race

African American recipients have poorer deceased donor graft survival compared to Caucasians[1]. This probably reflects multiple factors including higher incidence of DGF, higher incidence of acute and late acute rejection, stronger immune responsiveness, a predominantly Caucasian donor pool (with resultant poorer matching of HLA and non-HLA antigens), altered pharmacokinetics of immunosuppressive drugs, and a higher prevalence of hypertension. Socioeconomic factors associated with inability to pay for transplant medications, poorer access to high-quality medical care and noncompliance probably also play an important role.

Recipient Gender

Registry studies of the association of recipient gender with transplantation outcomes have yielded differing results. In the CTS database[2], female recipients had slightly better allograft survival than male recipients of deceased donor kidneys or HLA identical kidney[20]. An important difference between female and male transplantation candidates is the higher degree of sensitization of the former to HLA antigens and possible non-HLA antigens. Females tend to be more sensitized because of pregnancy and possible because of more blood transfusions related to menstruation.

Recipient Sensitization : before or after Transplantation

Patients who are broadly sensitized (e.g., panel reactive antibody [PRA] status >50%) at the time of transplantation generally have poorer early and late graft survival compared to nonsensitized recipients. This is mainly related to an increased incidence of complications in the early post-transplantation period such as DGF and acute rejection. The principal reasons for sensitization are previous transplants, pregnancy, and previous blood transfusions. Highly sensitized patients are often given more intensive immunosuppression to reduce the risk of rejection, but this also exposes them to risk of infection and malignancy.

There is accumulating evidence that the presence of donor specific and nondonor specific HLA antibodies are associated with inferior graft survival[21]. This evidence suggests that low grade antibody mediated rejection is an important cause of graft damage.

Recipient HCV Antibody and HBsAg

Recipients who are hepatitis C virus (HCV) antibody positive at the time of transplantation have poorer allograft survival and poorer survival[1,23]. Higher mortality rates appear to be related to infection and worsening liver disease[23]. Nevertheless, it seems that transplantation of selected HCV positive patients confers a survival benefit as opposed to remaining on the dialysis[26].

The adverse effects of hepatitis B virus (HBV) surface antigen positivity on post-transplantation outcomes are much less pronounced. This may in part reflect the better anti-HBV therapies available for transplant recipients that have been introduced in recent years.

Acute Rejection

Acute rejection has been consistently associated with an increased risk of graft loss. This is due to irreversible graft injury at the time of acute rejection and probably ongoing subclinical immunemediated injury. Such damage accentuates the effects of poor quality donor tissue, preoperative ischemic injury, nephron underdosing, and so forth. Acute rejection refractory to steroids, acute rejection where creatinine does not return near baseline, and late acute rejection (occurring after the first 6 months) are particularly associated with poorer graft and patient outcomes[17]. More severe histologic changes (e.g., Banff grade II or III cellular rejection) or severe acute antibody-mediated rejection are also associated with poorer graft survival. Although current immunosuppressive regimens have steadily decreased rates of acute rejection, this has not necessarily translated into a major improvement in long term graft survival.

Recipient Immunosuppression

Undoubtedly, the improvements in short and long term allograft survival reflect, in part, the effectiveness of the newer antirejection, drugs such as the cyclosporine, tacrolimus and mycophenolate mofetil. The short term improvements in allograft survival have been particularly impressive. The contribution of long term CNI therapy, particularly with currently used

maintenance doses, to chronic renal allograft dysfunction (and loss) remains controversial. The increases in short and long term graft survival in the CNI era (cyclosporine became widely used in the early 1980s) suggest that these antirejection effects override the nephrotoxic effects. Tacrolimus was more effective than cyclosporine in preventing acute rejection and allograft loss but at the expense of higher rates of diabetes mellitus[28].

There is limited evidence (registry data, not randomized trials) that mycophenolate mofetil improves long-term graft survival both by preventing overt acute rejection and possible by other mechanisms. Significant level of cyclosporine and tacrolimus produces 30% increase in bioavailability of mycophenalate mofetil. Short term studies of sirolimus have shown contradictory results[29,30]. In fact, one registry study suggests that sirolimus use is associated with inferior allograft survival[31].

Although antilymphocyte antibody preparations (e.g., antithymocyte globulin or interleukin-2 receptor blockers) are often used, particularly in the setting of DGF, their effects on long term graft survival have not been well studied. Recent United Network of Organ sharing data suggest that antibody induction protocols slightly reduce early acute rejection episodes in recipients with DGF and slightly improve graft survival. It is important to note that aggressive immunosuppression could adversely affect graft survival by promoting BK (polyoma) virus nephropathy or higher rates of death from opportunistic infections.

Recipient Compliance

Poor compliance with the immunosuppressive regimen is known to increase the risk of acute rejection, particularly late acute rejection, and chronic allograft dysfunction. The magnitude of this problem is difficult to define. In one study of patients followed up to 5 years after transplantation, 22.6% were identified as being noncompliant; this was associated with a large increased in risk of late acute rejection and of higher plasma creatinine[32].

Obesity

Obesity is increasingly common in Chronic Kidney Diseae-stage5 patients and is associated with more transplantation surgey-related complications, more DGF, higher mortality (related to cardiovascular complications), and poorer graft survival[33]. Similar evidence of poorer patients and graft outcomes has been reported by USDRS[1]. The poorer long term graft survival probably reflects the effects of DGF, nephron overwork, and more difficult dosing of immunosuppressive drugs. Nevertheless, most studies of patients with BMI >30 kg/m^2 suggest transplantation provides a survival benefit over remaining on the waiting list (on dialysis) at least up to a BMI of 41 kg/m^2.

Recipient Hypertension : Angiotensin system

Retrospective studies have shown that the greater the severity of post-transplantation hypertension is, the higher is the risk of graft loss[34]. Of course, hypertension could also be secondary to graft damage and not just a cause. No prospective human studies of the effect of treating hypertension on allograft

outcomes are available. However, control of hypertension is associated with improved allograft survival[35].

Multiple studies have confirmed the ability of angiotensin converting enzyme (ACE) inhibitors and angiotensin receptor blockers (ARBs) to slow the progression of both diabetic and nondiabetic proteinuric native kidney disease. ACE inhibitors and ARBs should be similarly beneficial in transplant kidney disease and thus should be used more frequently. While several studies have shown that both classes of drugs are effective in treating post-transplantation hypertension and reducing proteinuria in the short term, no long term studies of their effects on progression of transplant kidney dysfunction have been published. In one randomized, controlled trial, patients randomized to nifedipine had sustained improvement in GFR up to 2 years after transplantation; no improvement was seen in the lisinopril group. This may reflect the ability of nifedipine to attenuate CNI-induced vasoconstriction of the afferent arteriole[33].

Recipient Dyslipidemia

The prominence of the vascular lesions in CAN and the similarity of these lesions to atherosclerosis suggest that dyslipidemia plays a role in the pathogenesis of CAN and graft failure. Some studies have suggested that hypercholesterolemia and/or hypertriglyceridemia are associated with porer graft outcomes.

Recurrence of Primary Disease

Determining the incidence and prevalence of recurrent or de novo renal disease is difficult. The original cause of Chronic Kidney Diseae-stage5 is often unknown; most relevant studies are small and retrospective with variable follow-up periods. In one of the best performed studies of transplant recipients whose cause of Chronic Kidney Disease-5 was glomerulonephritis, the cumulative incidence of graft loss at 10 years was 8.4%[36]. Recurrence was the most important cause of loss, after chronic rejection and death. It is likely that a renal allograft survival continues to improve, recurrent or de novo disease will be increasingly diagnosed (both clinically and histologically) and will become a more important cause of late graft loss.

Proteinuria

The degree of proteinuria correlates with poorer renal outcome in both native and transplant kidney disease. Proteinuria may simply be a marker of renal damage, but there is speculation that proteinuria per se may accelerate allograft loss from CAN. ACE inhibitors and ARBs has definite role in showing the progression of proteinuria transplant renal disease.

Measures to improve renal allograft survival

- Increased living kidney donation : both related and nonrelated.
- Preemptive transplantation in live kidney transplantation.
- Increased donation from younger, previously healthy deceased donors.

- Preferential matching of younger deceased donors with younger recipients.
- Zero mismatching of HLA antigens
- Improved organ preservation
- Reduced cold ischemia time
- Nephron dosing (e.g. matching of donor recipient sex, body mass index)
- Calcineurin inhibitor sparing immunosuppressive protocols.
- Angiotensin converting enzyme inhibitors, angiotensin receptor blockers.
- Aggressive control of hyperlipidemia, hypertension.

MATERIALS AND METHODS

MATERIALS AND METHODS

Study place	**:**	Stanley Medical College Nephrology Department, Chennai
Study period	**:**	From October 2008 to April 2010
Study design	**:**	Prospective study
Study population	**:**	All 24 consecutive patients who underwent Cadaver transplant in this period were enrolled.

CRITERIA FOR TAKING UP FOR CADAVER TRANSPLANT:

- Patients in cadaver waiting list
- Patients with irreversible renal failure
- Dialysis dependent patients
- Patients under the age of 50 years
- Second Transplant patients
- ABO compatible patients

EXCLUSION CRITERIA:

- All Live related donor transplant and spousal transplant.
- Multi organ transplant
- Expanded criteria donor
- Non heart beating donor transplant
- Diabetic patients
- Patients with severe co-morbid conditions
- Patients with peripheral vascular disease

- Recipients demographic factors like Age, Gender, Occupation, Literacy were noted.
- Selection of recipients is based on their seniority in cadaver waiting list and cross match result.
- All recipients were maintained on Haemodialysis.
- All recipients were ABO compatible and cross-match negative and they are followed up regularly in our OP.
- Human Leukocyte Antigen (HLA) and Panel Reactive Antibody (PRA) were not done to any of our recipients.
- CMV status of the recipient was not checked routinely. However, if any suspicion of CMV infection like hepatitis, leucopenia, etc., the CMV status of the recipient was checked with pp65 antigen and treated with Vangancyclovir if they were positive.
- Graft survival was the primary outcome analysed.
- There were no drop outs from follow-up.
- Donor kidneys were received from various hospitals in Tamil Nadu and from our own hospital.
- Donors age ranged from 15 – 60 years without evidence of kidney disease or any infection.
- None of donors had diabetes mellitus or hypertension.
- All the donors had negative serology (HBV, HCV, HIV).

- ❖ All grafts were perfused with HTK solution (Custodial solution)
- ❖ Custodial (HTK) solution (in mmol/L)

• Sodium chloride	15.0
• Potassium chloride	9.0
• Potassium hydrogen 2-ketoglutarate	1.0
• Magnesium chloride	4.0
• Histidine Hcl	18.0
• Histidine	180.0
• Tryptophan	2.0
• Mannitol	30.0
• Calcium chloride	0.015

- ❖ They are stored in ice box with three bag technique during transportation
- ❖ Donor's age, sex, cause of death, graft side and abnormality and biochemical profile were noted.
- ❖ Transplant surgery was done by two teams of Urologists.
- ❖ Ethical Committee approval from Stanley Medical College, Chennai was obtained for this study.

DECEASED DONOR GRAFT ALLOCATION POLICY

A separate cadaver waiting list for each blood group of potential recipients is maintained according to their date of induction into haemodialysis. This seniority list is available online and it is supervised by transplant committee formed by the Government of Tamil Nadu.

Recipients with co–morbid conditions are temporarily deleted from the list and included again once they recover.

PROCEDURE

Pre operative treatment

All recipients were given Haemodialysis pre operatively. They were started on immunosuppression prior to surgery as below.

	Day before Surgery 4 p.m.	0 POD (4 a.m.)
T.Tacrolimus	**0.066 mg/kg**	**0.066 mg/kg**
T.MMF	**500 mg**	**500 mg**
T.Prednisolone	**0.5 mg/kg**	**0.5 mg/kg**

Operative Technique

Grafts are placed in the right iliac fossa after creating renal bed except in second transplant. Anastomosis of the renal vessels to the iliac vessels was performed as follows.

Graft artery to internal iliac artery (except one patient) – end to end.

Graft vein to external iliac vein – end to side.

Ureter anastomose to bladder obliquely in the region of the trigone. DJ stents were applied if required.

During anastomosis of graft vessels, methyl prednosolone 1 g was given as I.V. infusion.

Post operative treatment

Fluids (0.9% NS) were given according to their urine output. Immunosuppression was given as follows:

T.Tacrolimus 0.066 mg/kg Bd (Target tacro level 10 – 12 ng/ml subsequently reduced to 5ng/ml by 6 months)

T.MMF 500 mg Bd

T.Prednisolone 0.5 mg od

Tacrolimus levels were assessed on POD-5 for all recipients. Doppler of graft vessels are assessed on POD-7. Recipients urinary Foley's Catheter was removed on POD-7. Drainage tube was removed if drainage fluid is less than 50 ml. DJ stent was removed on 4^{th} post operative week.

After 10 days, recipients were discharged and they were seen as outpatient at intervals of twice weekly for one month than weekly once for two months, thereafter fortnightly for one year and monthly for one year life long. During each visit, patient's condition, renal function test and complete blood count were analyzed. Post operative drugs including immunosuppressants are given free of cost and all investigations are done at no cost.

STATISTICAL METHODOLOGY

The statistical analysis has been done by using SPSS (Statistical Package on Social Science) version 10.0 has been used.

The non-parametric model can be used to find out the relationship of categorical variable. One of the methods is Fisher's exact Chi-square. It can be used when the cell counts are less than five. Here for all the tables the cell value is less than five. Hence it has been used.

To find out the relation of Graft function with other risk factors like age, gender, creatinine in one month, creatinine in 6 month, dialysis duration, LVH, Intra operative status, Postoperative status, CIT, and Transplant order, the above method has been used.

The factors like age, BMI, creatinine in one month, creatinine in six months, has been classified or categorized into two groups according to their mean or average. The other factors like dialysis duration and Cold Ischemia Time has been classified or categorized into two groups according to median value.

RESULTS

RESULTS

24 patients received cadaver graft in our center from October 2008 to April 2010. Mean age of the recipients was 33.8 years (±SD 7.17). Among them males were 20(83%) and female were 4(17%).

Out of 24 patients one had ADPKD, one had biopsy proven IgA nephropathy and the remaining had contracted kidney for which native kidney biopsy was not done. The cause of chronic kidney disease for them is not known.

During the period of study, Diabetic patients were not included in the cadaver waiting list. All recipients were on antihypertensives. One of the recipients was Hepatitis B positive. One was Hepatitis C positive. None of the patients received induction therapy like ATG (Anti thymocyte globulin), Daclizumab or Basiliximab.

Among the recipients 20(83.3%) were males and 4(16.7%) were females. Only one recipient had second transplant and all other had first transplant. One patient had ADPKD, one patient had biopsy proven IgA nephropathy and all other had unknown etiology. All of them had normal renal and iliac vessels (doppler done pre operatively). All the recipients received tacrolimus, mycophenolate mofetil and prednisolone

Among the recipients 8 were of Blood group O positive(33.3%), 10 were B positive(41.7%), 3 were AB positive(12.55%), one each of A positive(4.2%), A negative(4.2%) and B negative(4.2%).

Average age of the donors was 32.3 years (±SD14.32%). Among them males were 16(66.7%) females were 8 (33.3%). 3 (12.6%)donors had fall from height as the cause of brain death. Others were due to road traffic accident.

Among the donors 9(37.55) were O positive 10(41.7%) were B positive, 3(12.5%) were A positive, 2(8.3%) were AB positive.

Among the received grafts 17(70.8%) were left sided graft, 7(29.2%) were right sided graft. 17(70.8%) graft were without any vessel or ureteric anomalies. Among remaining grafts 4(16.7%) had 2 renal arteries, 1 (4.2%) had 2 renal veins, 2(8.4%) had 3 renal arteries. All the grafts were perfused with HTK (custodial) solution.

Out of total 24 recipients, 9(37.5%) had DGF, 8(33.3%) died and one had graft nephrectomy .

Intraoperatively, 17(70.8%) recipients did not have any intraoperative events, one (4.2%) recipient had bleeding from renal bed, two (8.3%) had hypotension during surgery, one (4.2%) had on the table mottling of graft after clamp release and one (4.2%) had graft artery anastomized close to the hilum.

Postoperatively, two (8.4%) recipients had sepsis and one (4.2%) had culture proven fungal sinusitis, one (4.2%) had pancreatitis, one (4.2%) had biopsy proven ATN and one (4.2%) had ischemic necrosis of the right leg.

The influence of various demographic, biochemical and clinical parameters of recipients and donors in influencing graft function are analyzed.

Only two factors are statistically significant to influence the graft function. Cold ischemic time significantly influenced the graft function and creatinine at one month predicts the graft survival.

DISCUSSION

DISCUSSION

Transplant failure represents a current challenge in nephrology. In this study prolonged cold ischemic time was found to affect graft survival significantly. One month creatinine value predicted patient's survival. DGF occured among 37.5% of deceased graft recipients. Compared to western studies (90%), patient survival was 66.7% and graft survival was 62.5% only. This may be because of the learning curve in cadaver transplants in spite of around 40 live transplants being done every year. Difficulty in deceased donor graft procurement, transportation of graft, delay in getting cross match results especially during odd hours and different transplant surgeons contributed to the poor graft survival. Over a period of time these are bound to improve.

In this study the prolonged cold ischemic time is mainly attributed to transplant being performed the following day, when the graft is received at odd hours.

Infection was found to be the prime cause of death in deceased donor graft recipients. Out of 8 deaths, 3 were due to sepsis, one each due to biopsy proven acute rejection, HUS and CAN.

The graft survival was not significantly influenced by recipient's age, gender, dialysis duration, intra-operative hypotension, post – operative sepsis and their serology status. The number of cadaver transplants may not be sufficient to discern statistically significant trend.

THE CLINICAL COURSE OF PATIENTS WHO EXPIRED AND A PATIENT WHO UNDERWENT GRAFT NEPHRECTOMY

CASE 1: Kondia Raj

Male aged 36 years presented with uremia and contracted kidneys. During the surgery the renal graft was found to have triple renal artery with Carrel aortic patch. He had a uneventful post operative period. But later he developed graft dysfunction after 15 months. Graft biopsy showed CAN. Then he become dialysis dependant and died at his native palace.

CASE 2: Bala Raman

Male, aged 32 years had been uremic for one year. He had uneventful intraoperative and postoperative period. He was maintaining normal graft function for almost one year post transplant. Then he developed acute hepatitis due to HCV virus and hepatic encephalopathy from which he did not recover and died.

CASE 3: Sasi Kumar

Male aged 29 years, a case of CKD stage-5 of unknown etiology on maintenance haemodialysis for two years. He had uneventful intraoperative and postoperative period. He was maintaining normal graft function for three months. Then he developed CMV pneumonia and died with normal graft function.

CASE 4 : Dasan

Male aged 48 years, a known case of ADPKD and positive serology for hepatitis B virus on maintenance haemodialysis for two years. He had uneventful intraoperative period and he developed fever postoperatively on 14^{th} day from which he recovered with antibiotic. He developed DGF post operatively and

dialyzed eight times. His discharge creatinine was 2.4. After three months he developed bronchopneumonia, sepsis and respiratory failure. He was on ventilator for one day and died.

CASE 5 : Xavier

Male aged 43 years on maintenance haemodialysis for two years received deceased donor graft with two renal arteries. Main renal artery anastomosed to internal iliac artery and accessory artery to external iliac artery. Following clamp release, his right leg pulse was not palpable. He was reexplored and no thrombus was found. The graft was reanastomosed. Next day patient developed ischemic necrosis of right leg due to femoral artery thrombus for which he underwent femoro femoral bypass. The same day he expired due to sepsis and arrhythmia.

CASE 6 : Basker

Male aged 38 years, a case of CKD of unknown etiology on maintenance haemodialysis for three months. He had uneventful intraoperative and postoperative period and he was maintaining normal graft function for 4 month post transplant. Then he developed graft dysfunction and returned to dialysis. Graft biopsy showed features of HUS. Subsequently, he became HCV positive and died in outside hospital.

CASE 7 : Eswaran

Male aged 31 years on maintenance haemodialysis for one year. He underwent second transplant and he had persistent hypotension intraoperatively. He returned to dialysis due to DGF. He had persistent blood stained drainage in DT for which he was reexplored and blood clots found around graft which was

evacuated and graft found to be normal. Open graft biopsy done showed features of both acute humoral and cellular rejection. He was not treated for that as he had persistent fever then he developed pain over graft site and graft swelling. Graft nephrectomy done following which patient expired due to sepsis.

CASE 8 : Rajan

Male aged 36 years on maintenance haemodialysis for three months had uneventful transplantation. He was returned to dialysis due to DGF and found to have fungal sinusitis and a palate swelling. Culture of palate lesion grew mixed organism of herpes, Candida and mucor. Then he developed CNS infection and died.

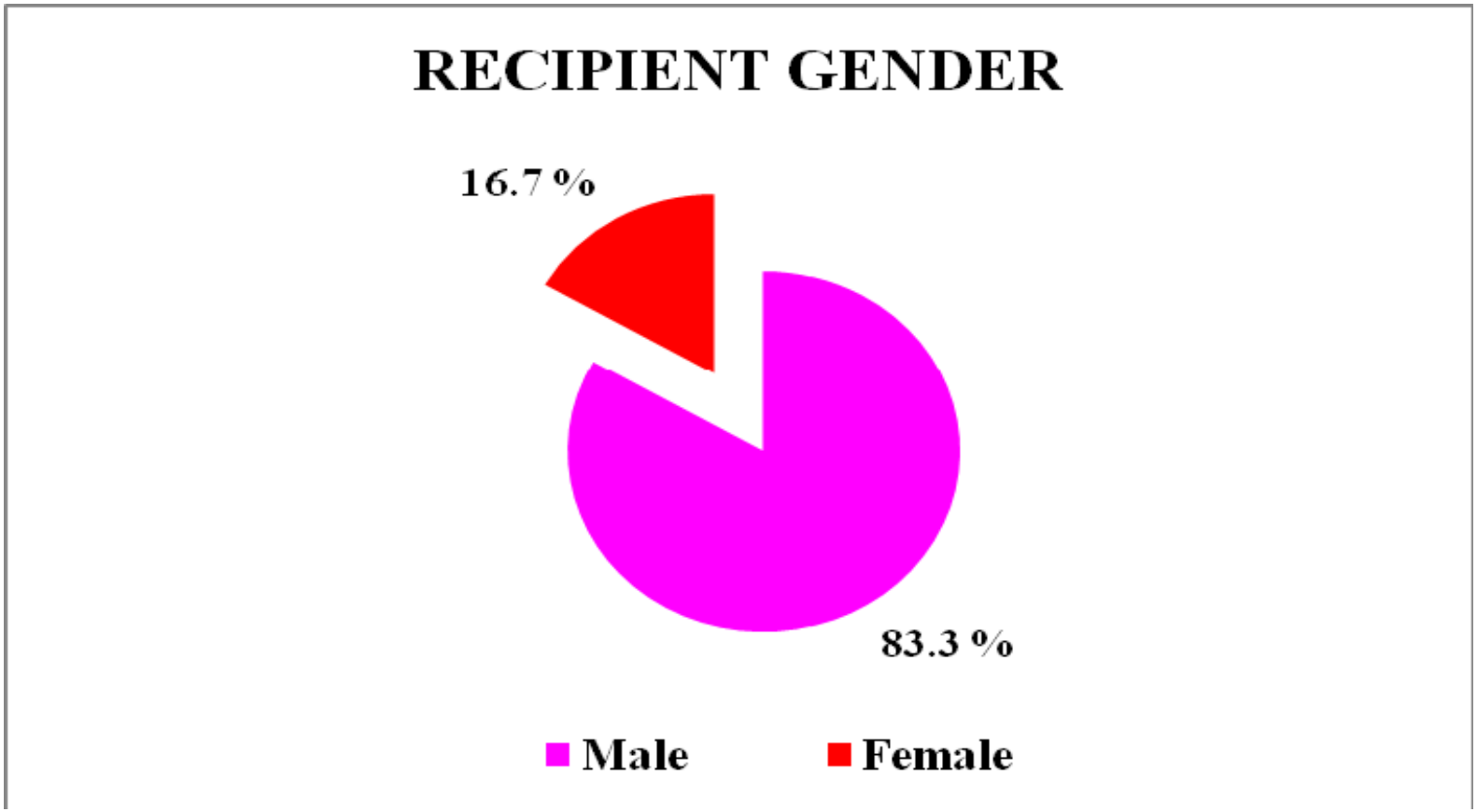
CASE 9 : Sakthivel

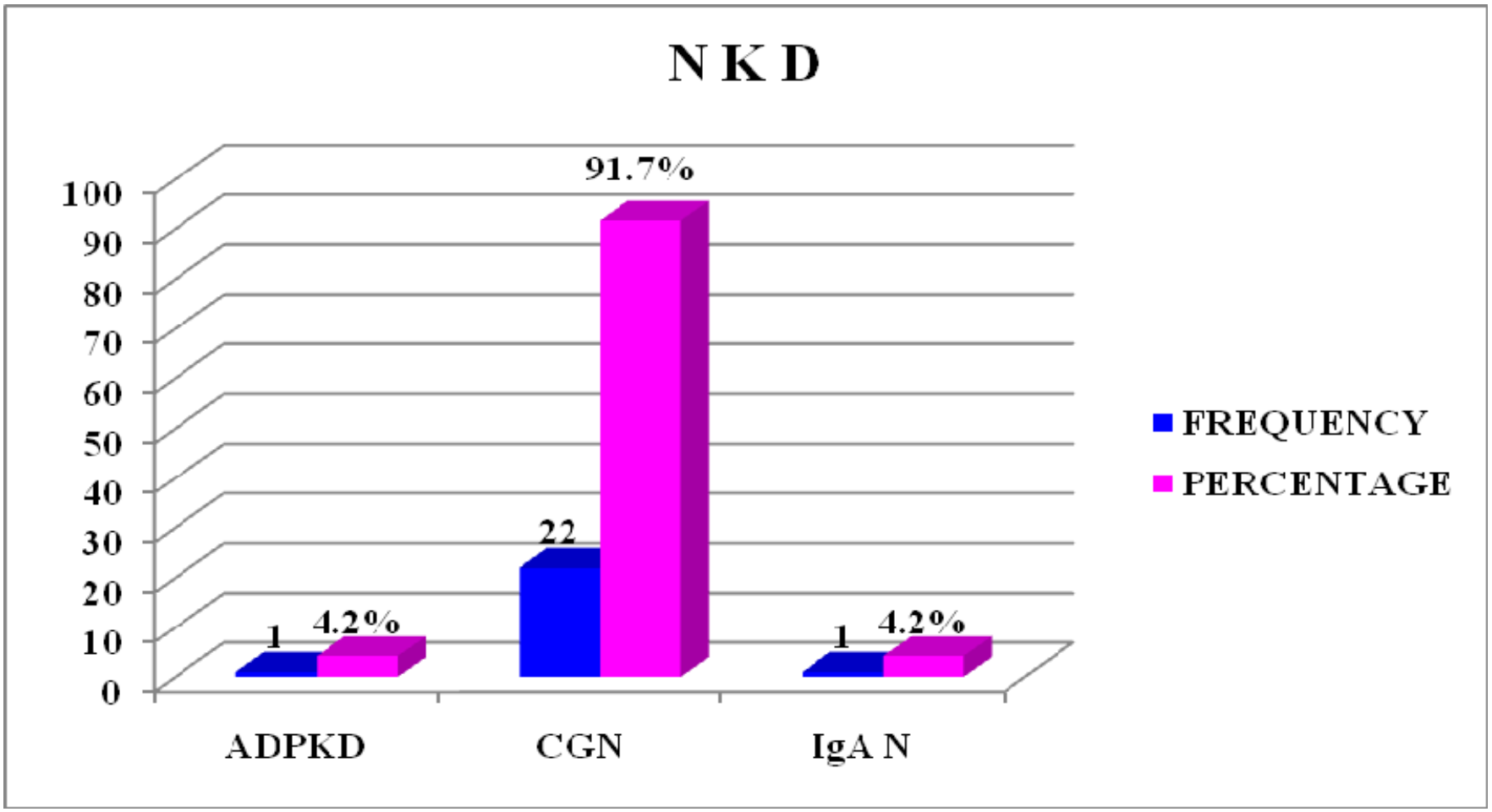
Male aged 22 years on maintenance haemodialysis for three year. He is a known case of hepatitis C positive. During surgery after clamp release graft become mottled on table. After 30 minutes kidney become pink and abdomen closed. Postoperatively he developed DGF and returned to dialysis. Two weeks later he developed hypotension and abdomen swelling. He was taken up for graft nephrectomy. He was found to have graft rupture. Graft biopsy showed features of severe ATN. He is on regular haemodialysis and he is alive and healthy.

TABLE - 1

BASELINE CHARACTERISTICS OF RECIPIENTS

No	Characteristics	Frequency	Percentage
1	**Gender**		
	Male	20	83.3
	Female	4	16.7
	HT	24	100
2	**Blood Group**		
	O positive	8	33.3
	B positive	10	41.7
	A positive	1	4.2
	AB positive	3	12.5
	B Negative	1	4.2
	A Negative	1	4.2
3	**NKD**		
	ADPKD	1	4.2
	Unknown cause	22	91.7
	IgA N	1	4.2
4	Normal Doppler	24	100
5	**Graft side**		
	Left	17	70.8
	Right	7	29.2
6	Anomaly	7	29.2
7	**I/II Transplant**		
	I	23	95.8
	II	1	4.1
8	ECHO	10	41.7
9	**Immunosuppression**		
	Tacro + MMF+ Prednisolone	24	100


RECIPIENT GENDER
16.7 %
83.3 %
Male
Female


N K D
100
90
80
70
60
50
40
30
20
10
0
91.7%
22
1
4.2%
1
4.2%
ADPKD
CGN
IgA N
FREQUENCY
PERCENTAGE

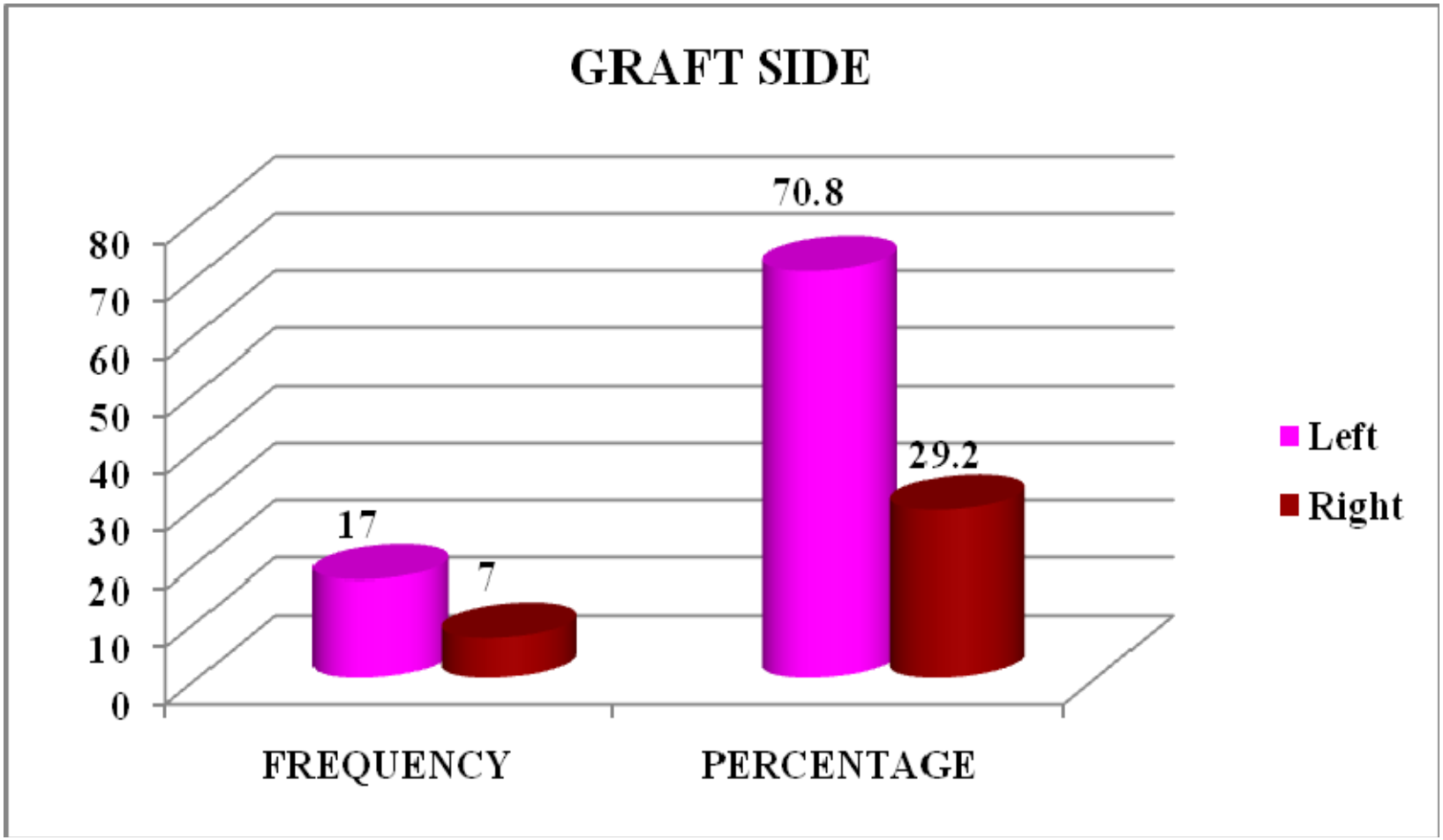
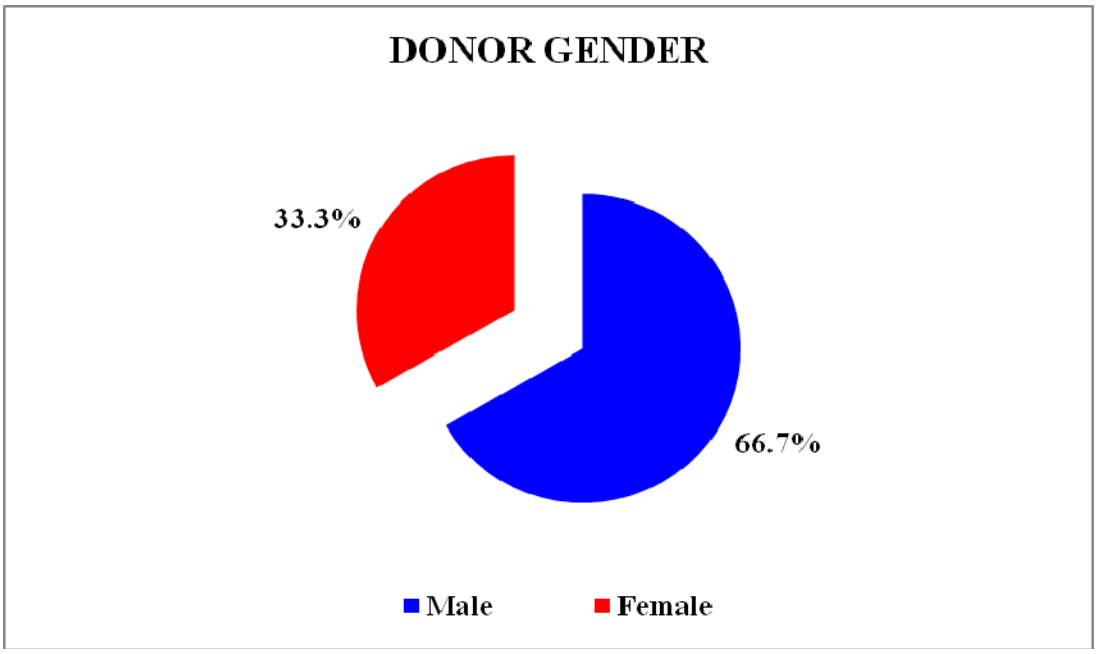

GRAFT SIDE
80
70
60
50
40
30
20
10
0
17
7
70.8
29.2
FREQUENCY
PERCENTAGE
Left
Right

TABLE- 2

BASELINE CHARACTERISTICS OF DONOR

No.	Characteristics	Frequency	Percentage
1	**Gender**		
	Male	16	66.7
	Female	8	33.3
2	**Blood Group**		
	O positive	9	37.5
	B positive	10	41.7
	A positive	3	12.5
	AB positive	2	8.3
3	**Cause of Death**		
	RTA	21	87.5
	Fall from Height	3	12.5



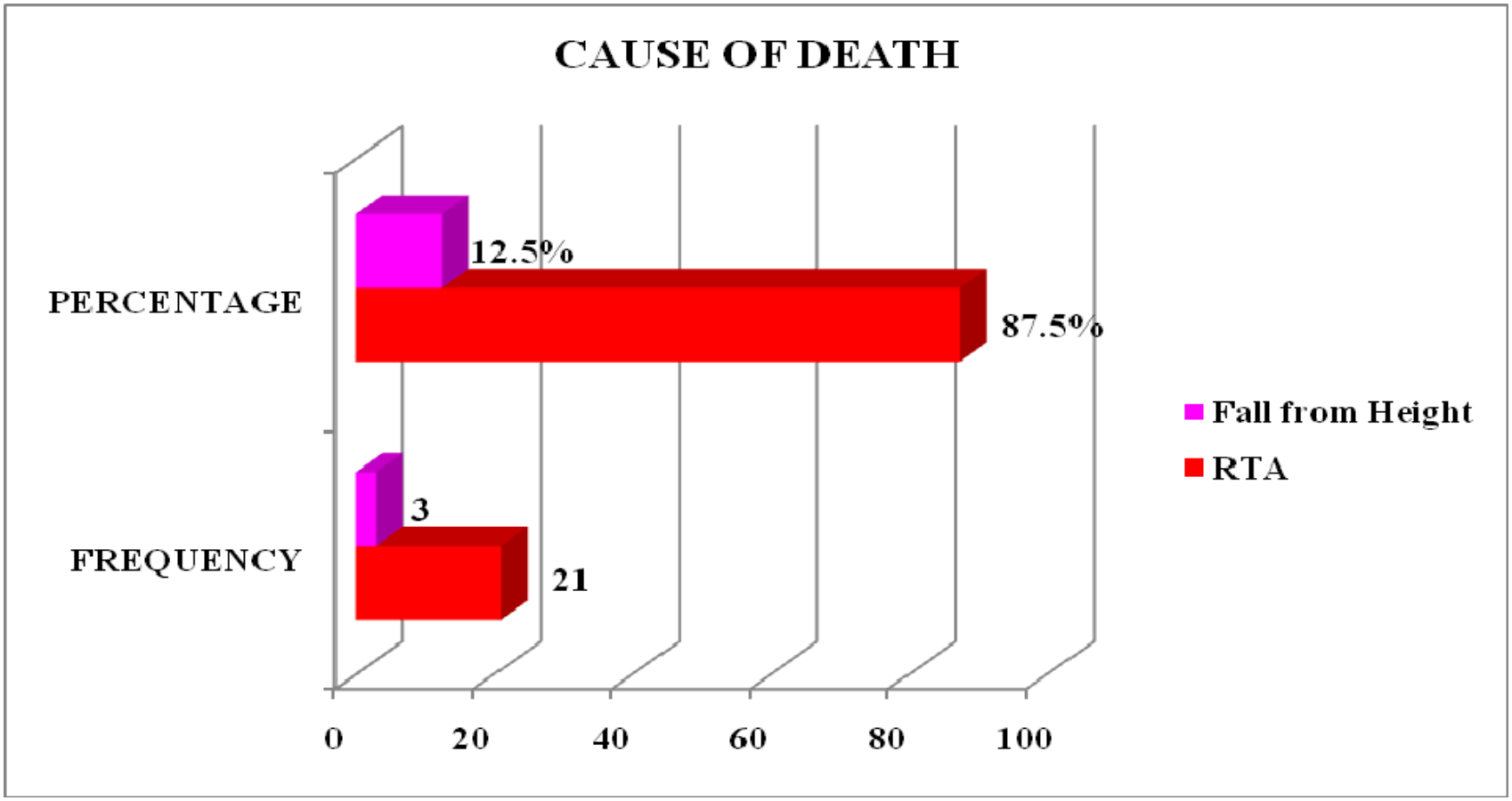

CAUSE OF DEATH
PERCENTAGE
FREQUENCY
12.5%
87.5%
3
21
0
20
40
60
80
100
Fall from Height
RTA

TABLE 3

PATIENT SURVIVAL

Outcome	Frequency	Percentage
Death	8	33.3
Alive	16	66.7

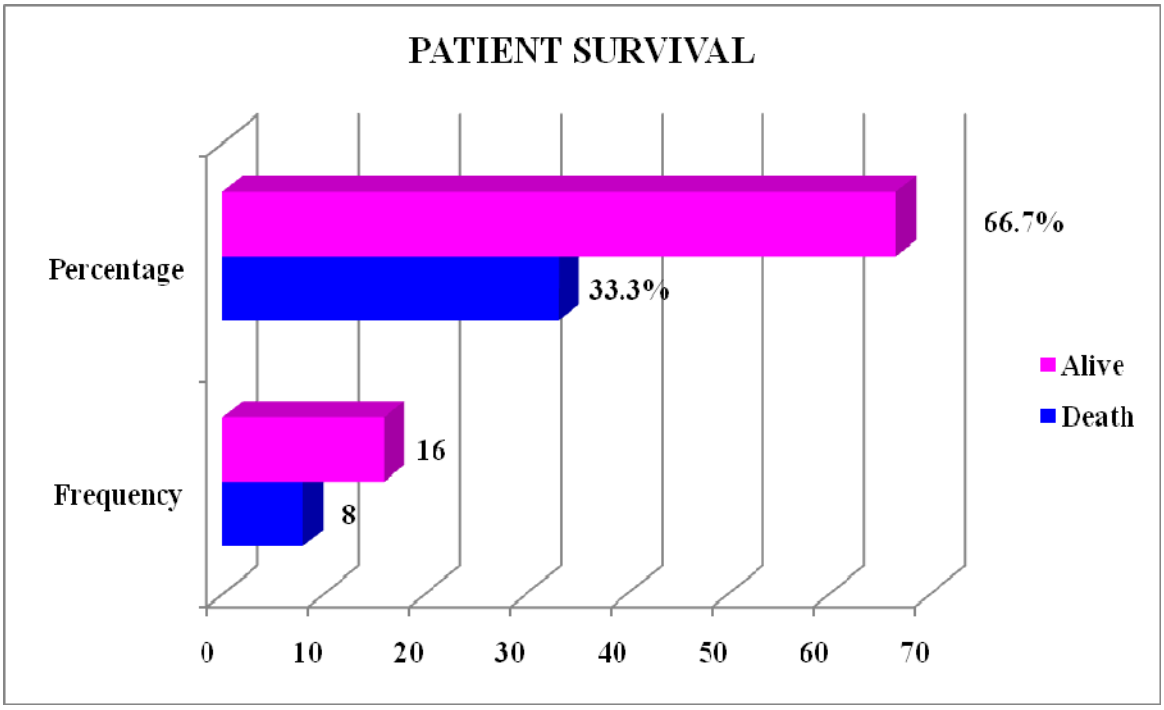


TABLE 4

GRAFT SURVIVAL

Outcome	Frequency	Percentage
DGF	9	37.5
Normal Function	15	62.5

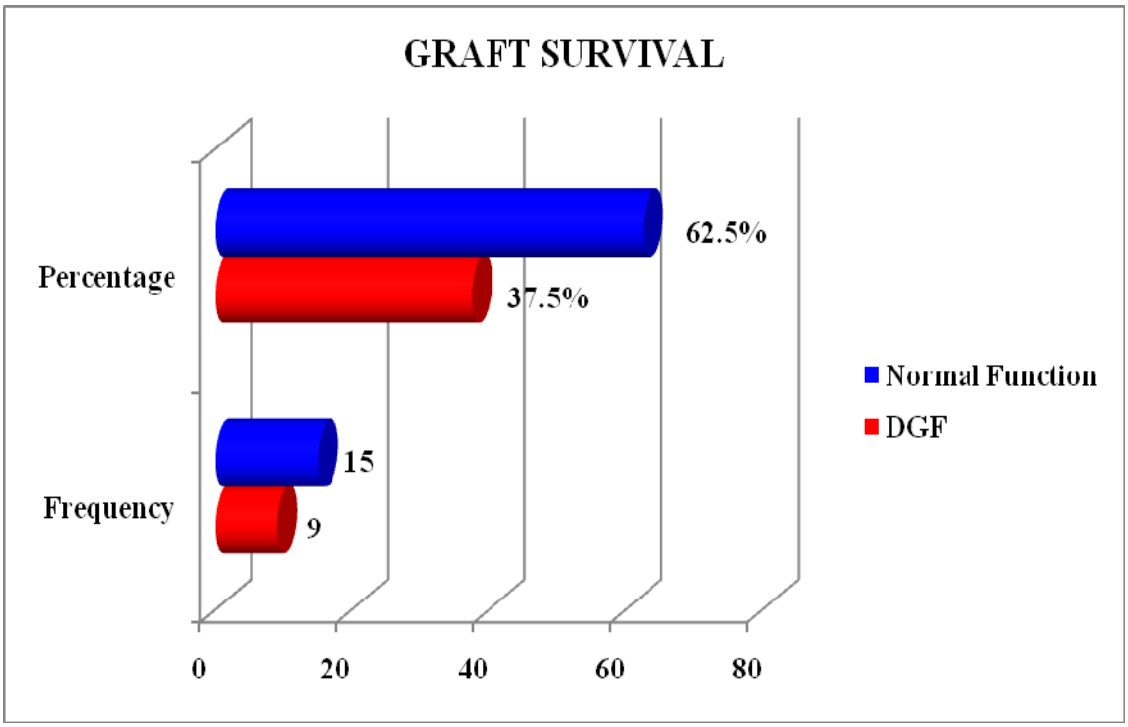


TABLE 5

INTRA OPERATIVE EVENTS

No	Intra Operative Events	Frequency	Percentage
1.	Bleeding from renal bed	1	4.2
2.	External Iliac – Anastamosis	1	4.2
3.	Hilum – Anastamosis	1	4.2
4.	Hypotension	2	8.3
5.	Mottling	1	4.2
6.	Uneventful	17	70.8
7.	Venous Leak	1	4.2

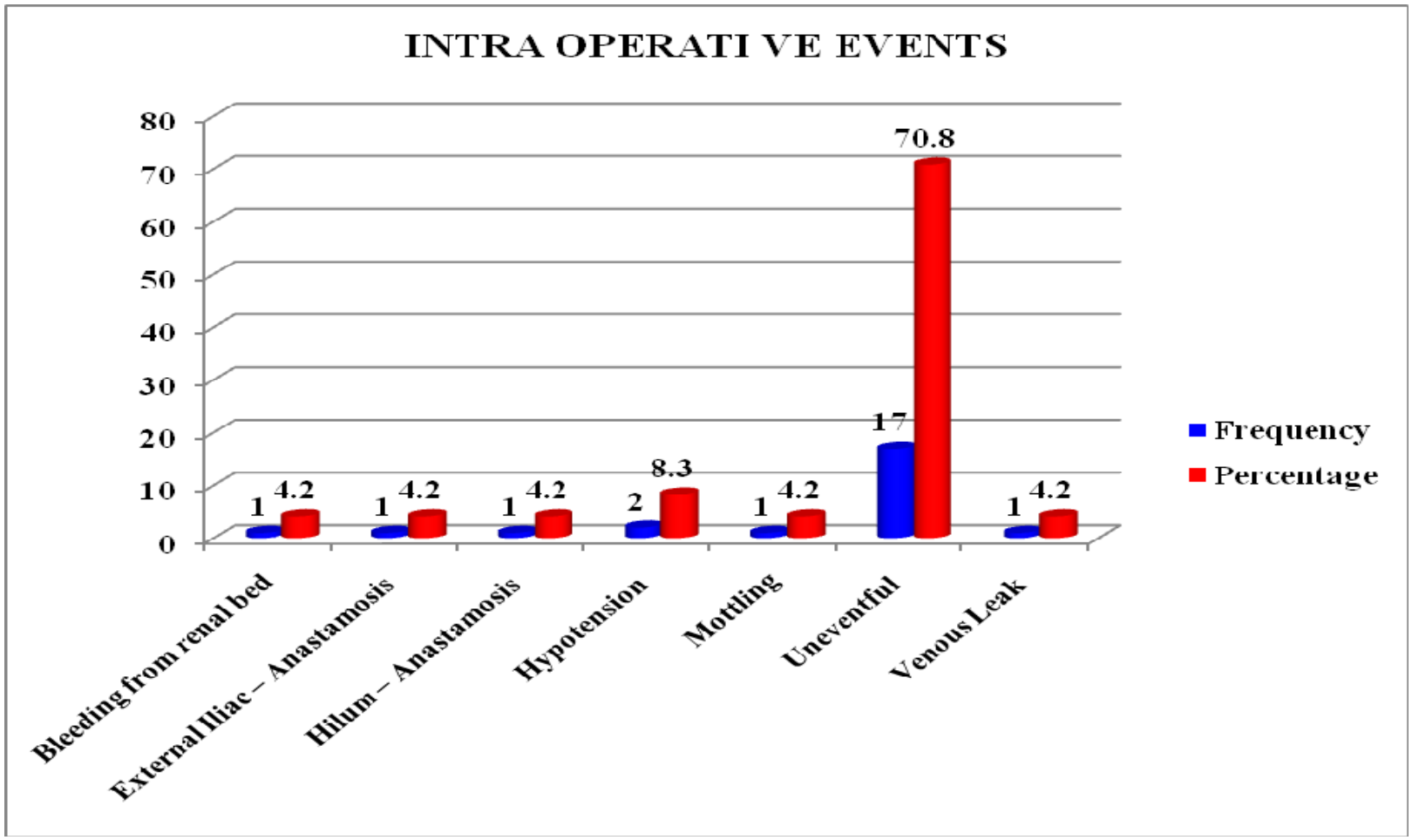

INTRA OPERATI VE EVENTS
80
70
60
50
40
30
20
10
0
1
4.2
1
4.2
1
4.2
2
8.3
1
4.2
17
70.8
1
4.2
Bleeding from renal bed
External Iliac – Anastamosis
Hilum – Anastamosis
Hypotension
Mottling
Uneventful
Venous Leak
Frequency
Percentage

TABLE 6

POST OPERATIVE EVENTS 0 TO 14 DAYS

No.	Post Operative Events	Frequency	Percentage
1.	Accelerated hypertension	1	4.2
2	Biopsy proven ATN	1	4.2
3	Fungal Sinusitis	1	4.2
4	Hypotension	1	4.2
5	Ionotropic requirement	1	4.2
6	Nephrectomy	1	4.2
7	Nil Events	11	45.8
8	Pancreatits	1	4.2
9	Right Leg Ischemia	1	4.2
10	Sepsis	2	8.3
11	Stitch Abcess	1	4.2

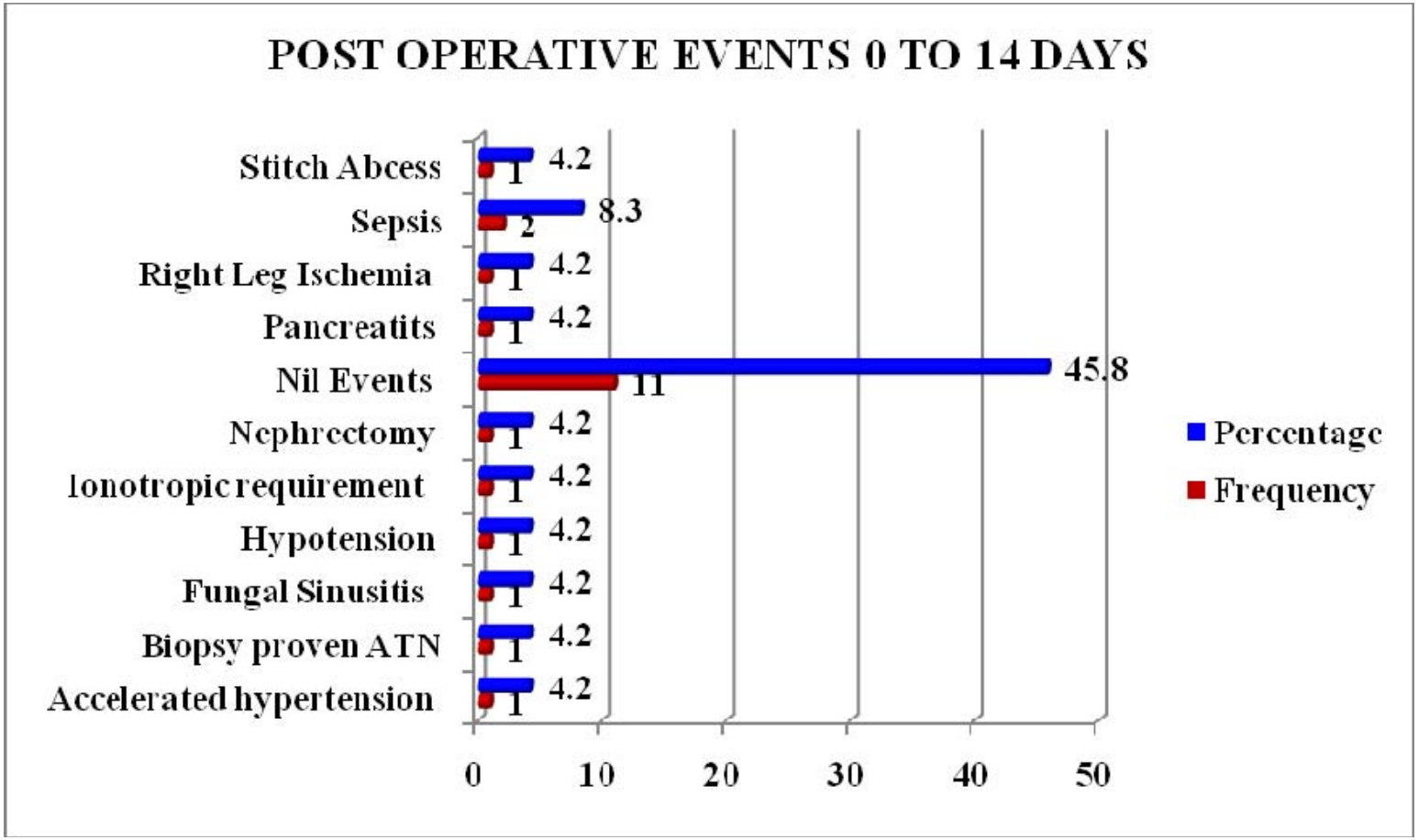
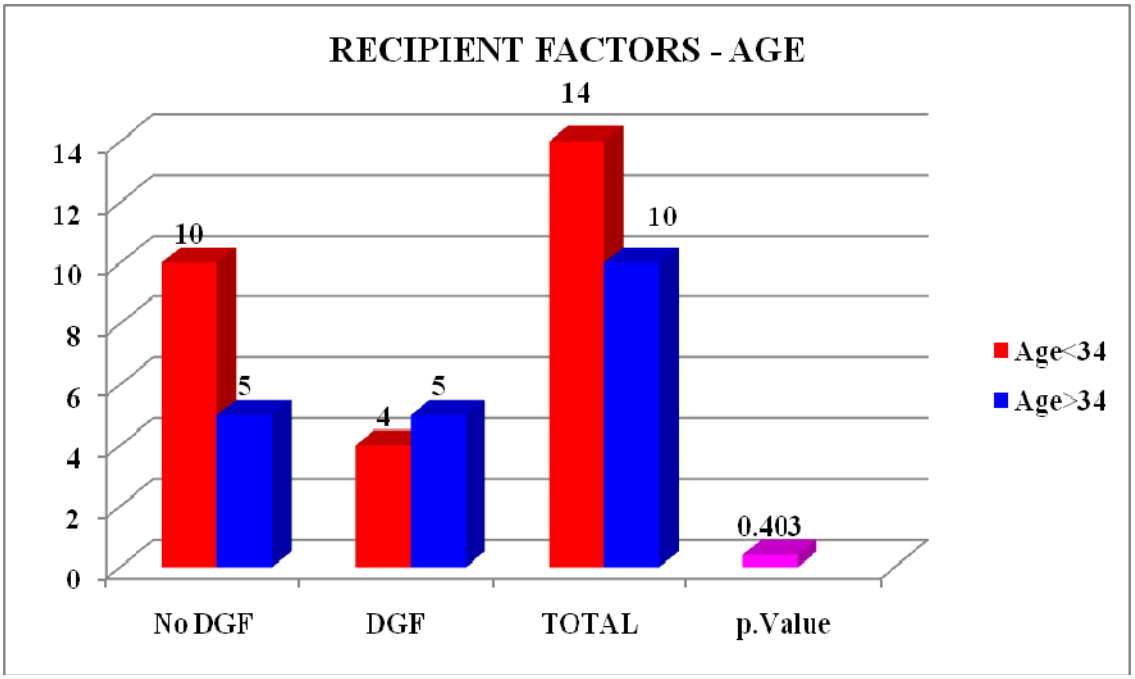
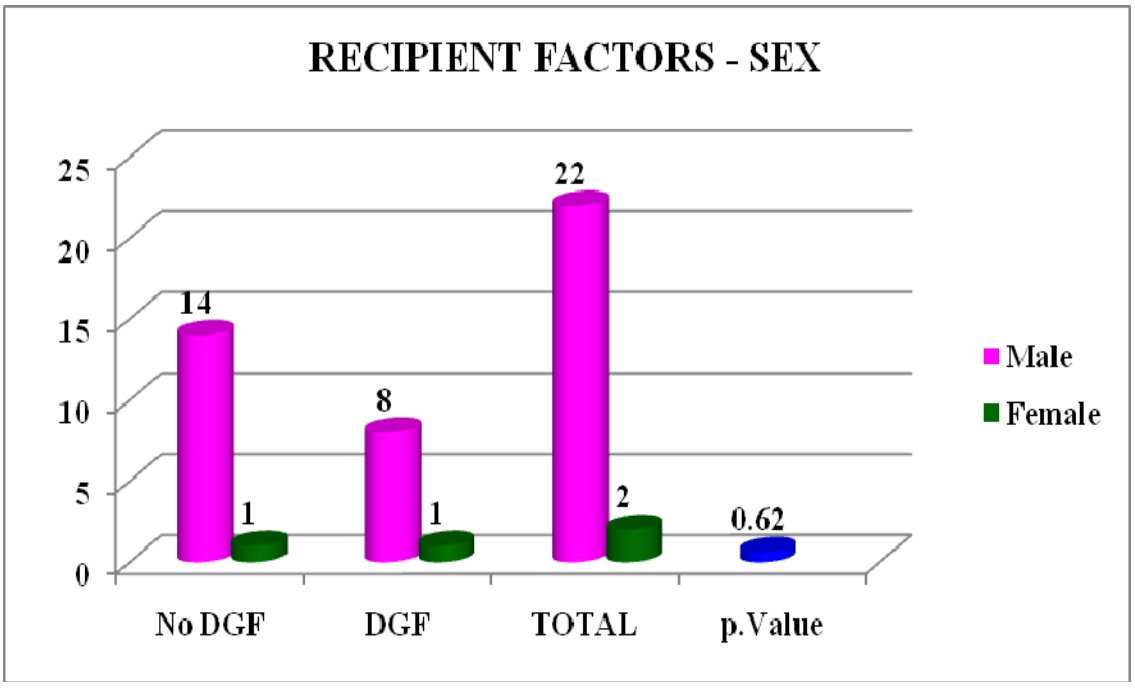

POST OPERATIVE EVENTS 0 TO 14 DAYS
Stitch Abcess
Sepsis
Right Leg Ischemia
Pancreatits
Nil Events
Nephrectomy
Ionotropic requirement
Hypotension
Fungal Sinusitis
Biopsy proven ATN
Accelerated hypertension
4.2
8.3
4.2
4.2
45.8
4.2
4.2
4.2
4.2
4.2
4.2
1
2
1
1
11
1
1
1
1
1
1
0
10
20
30
40
50
Percentage
Frequency

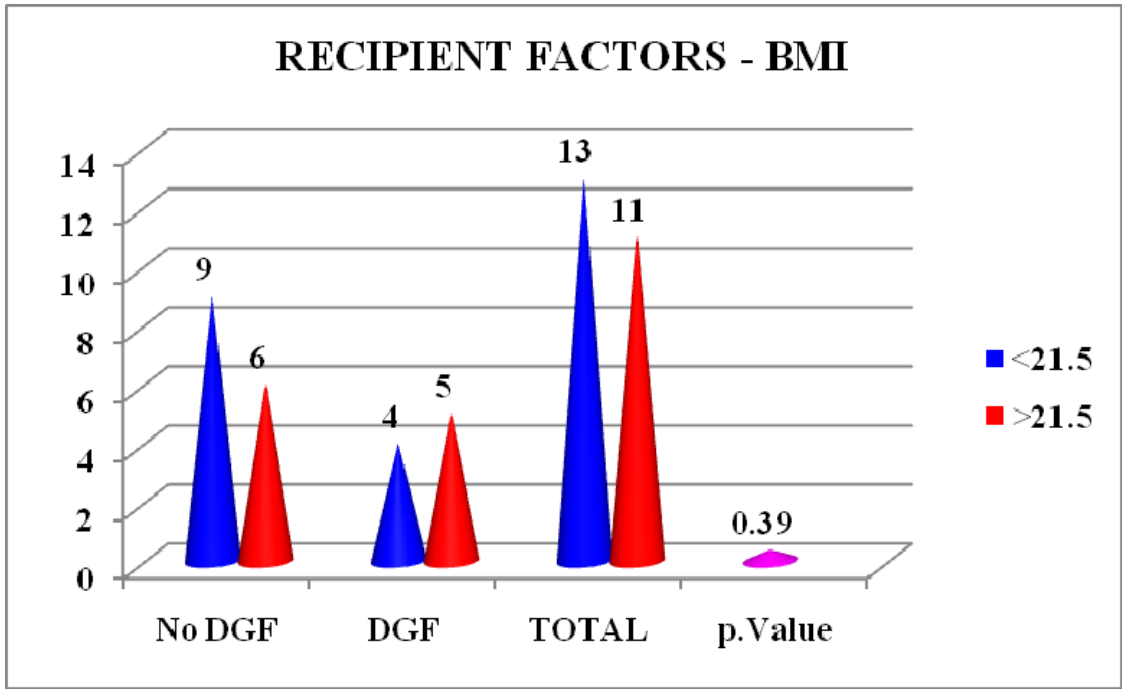
TABLE 7

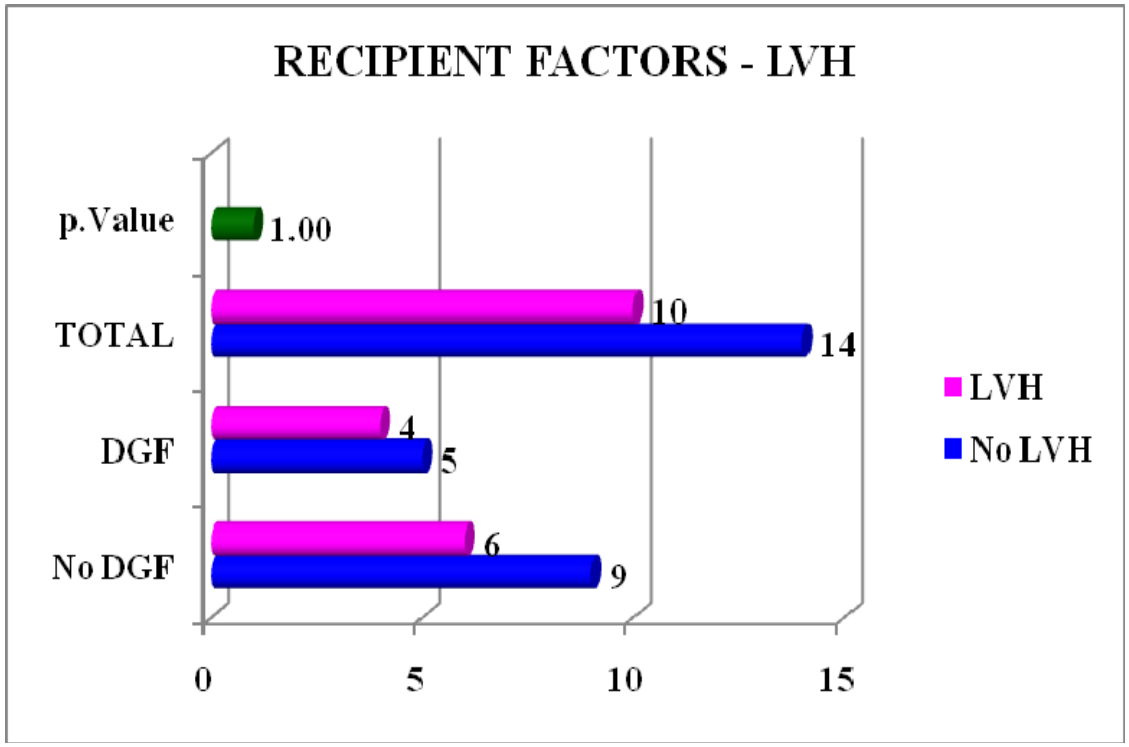
RECIPIENT FACTORS

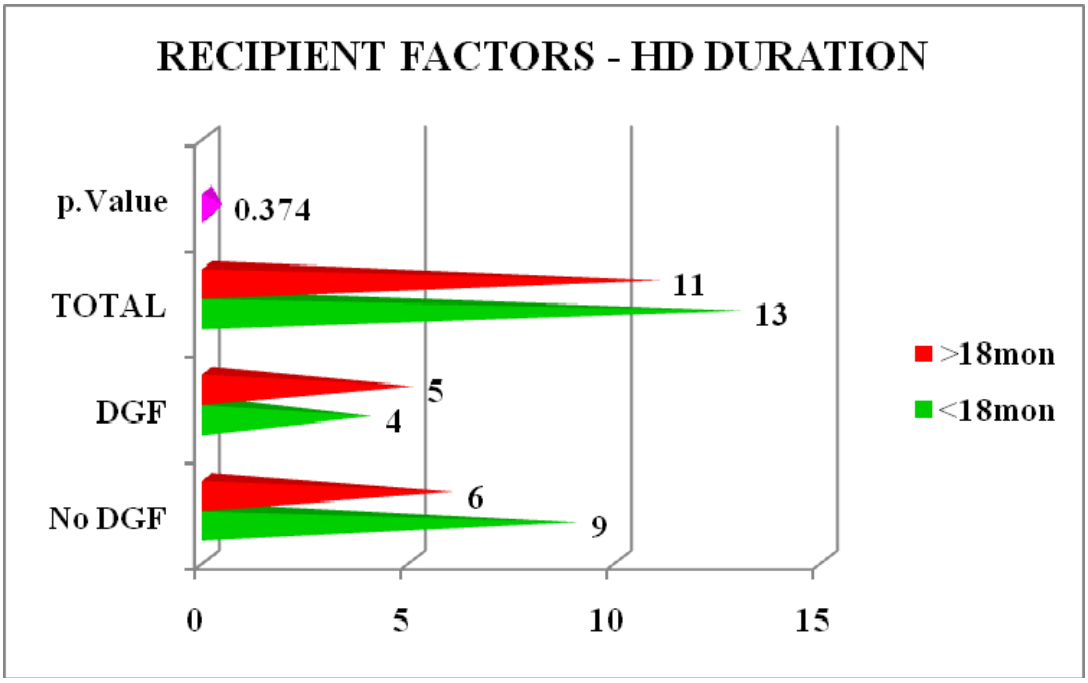
Variable	No DGF	DGF	TOTAL	P Value
Age				
Age<34	10	4	14	0.403
Age>34	5	5	10	
Sex				
Male	14	8	22	0.620
Female	1	1	2	
BMI				
<21.5	9	4	13	0.390
>21.5	6	5	11	
Tx I/II				
I Tx	15	8	23	0.375
II Tx	0	1	1	
LVH				
No LVH	9	5	14	1.00
LVH	6	4	10	
HD-Duration				
<18mon	9	4	13	0.374
>18mon	6	5	11	
HBV/HCV				
NEGATIVE	15	7	22	0.673
POSITIVE	0	2	2	


RECIPIENT FACTORS - AGE
14
12
10
8
6
4
2
0
10
5
4
5
14
10
0.403
No DGF
DGF
TOTAL
p.Value
Age<34
Age>34


RECIPIENT FACTORS - SEX
25
20
15
10
5
0
14
1
8
1
22
2
0.62
No DGF
DGF
TOTAL
p.Value
Male
Female


RECIPIENT FACTORS - BMI
14
12
10
8
6
4
2
0
9
6
4
5
13
11
0.39
No DGF
DGF
TOTAL
p.Value
<21.5
>21.5


RECIPIENT FACTORS - LVH
p.Value
TOTAL
DGF
No DGF
1.00
10
14
4
5
6
9
0
5
10
15
LVH
No LVH


RECIPIENT FACTORS - HD DURATION
p.Value
TOTAL
DGF
No DGF
0.374
11
13
5
4
6
9
0
5
10
15
>18mon
<18mon

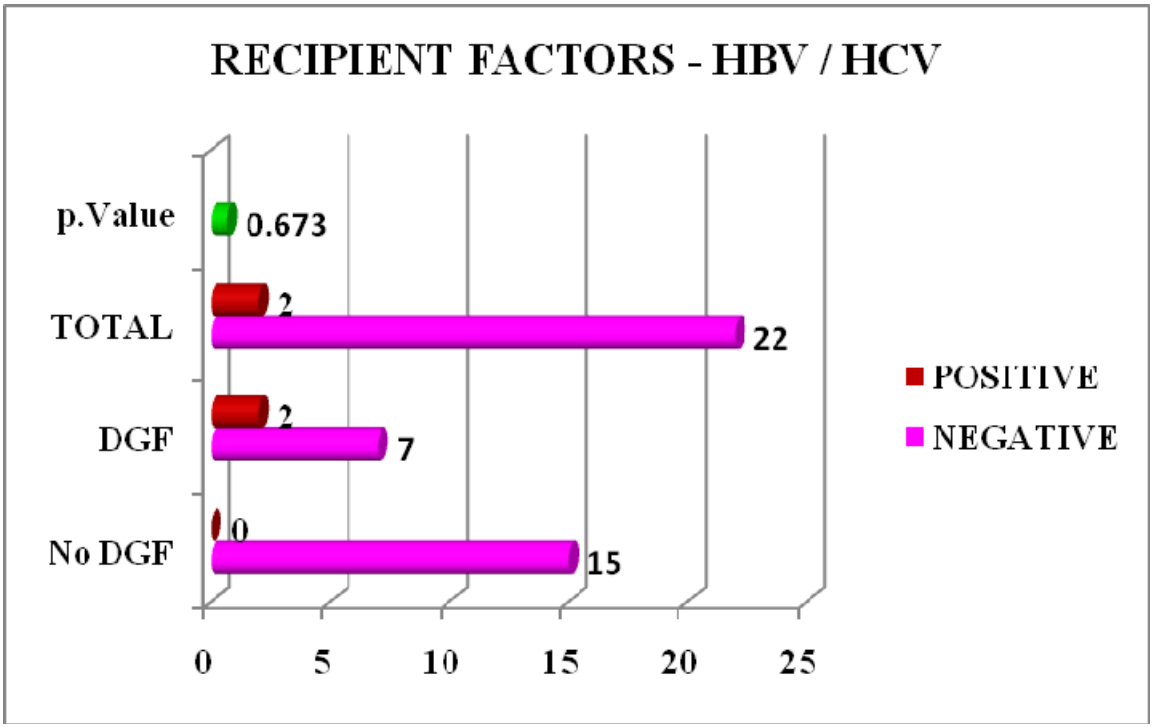
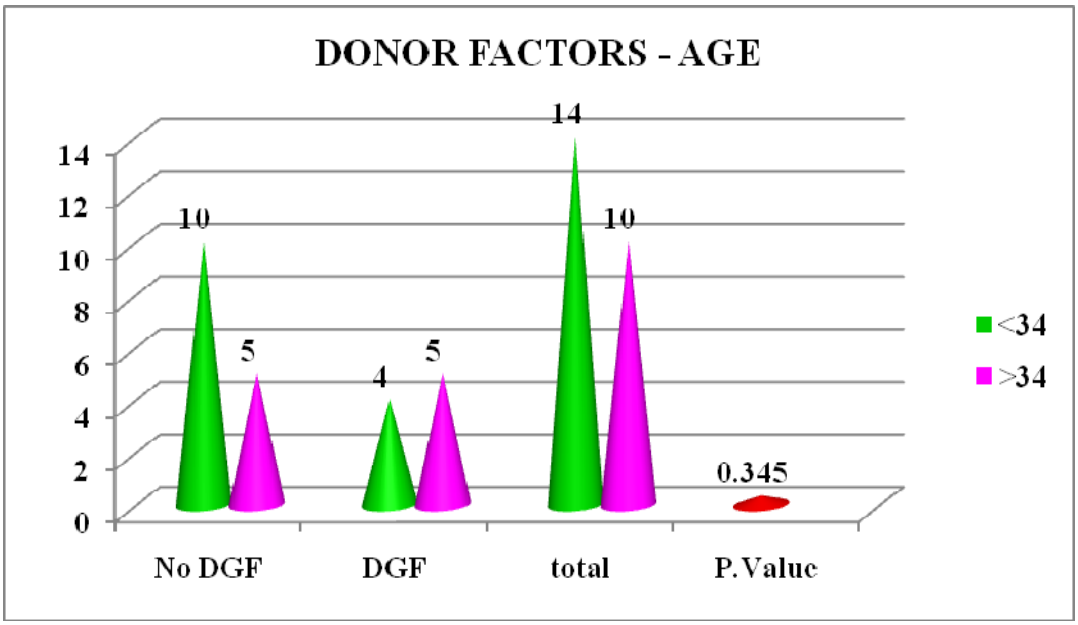

RECIPIENT FACTORS - HBV / HCV
p.Value
TOTAL
DGF
No DGF
0.673
2
22
2
7
0
15
0
5
10
15
20
25
POSITIVE
NEGATIVE

TABLE 8

DONOR FACTORS

Variable	No DGF	DGF	total	P Value
Age				
<34	10	4	14	0.345
>34	5	5	10	
Gender				
Male	10	7	17	0.669
Female	5	2	7	



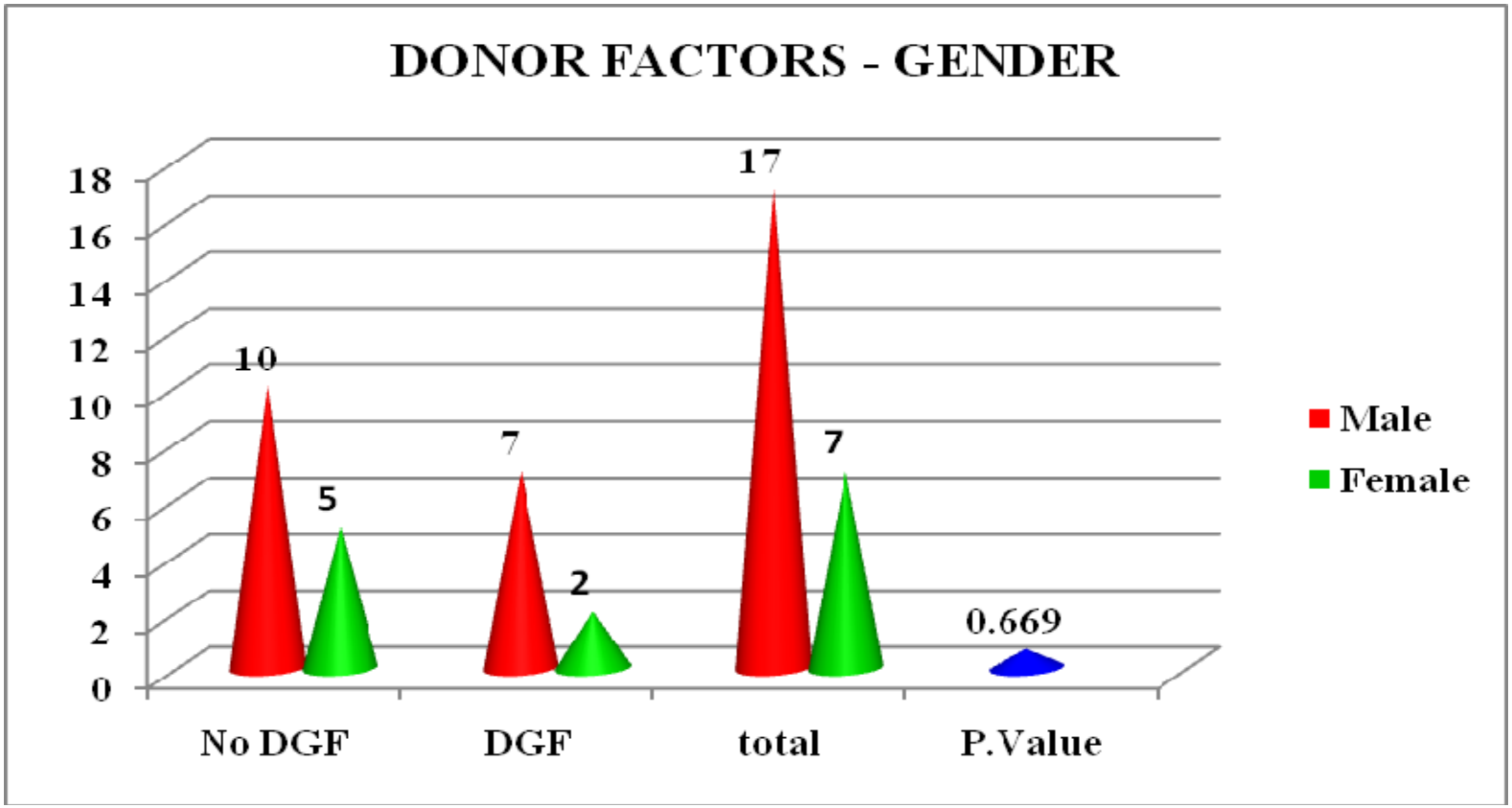

DONOR FACTORS - GENDER
18
16
14
12
10
8
6
4
2
0
10
5
7
2
17
7
0.669
No DGF
DGF
total
P.Value
Male
Female

TABLE 9

INTRA OPERATIVE FACTORS

Variable	No DGF	DGF	TOTAL	P Value
Hypotension				
YES	15	7	22	0.130
NO	0	2	2	
CIT				
<8 Hrs	7	0	7	
>8 Hrs	8	9	17	0.020★

★-Significant

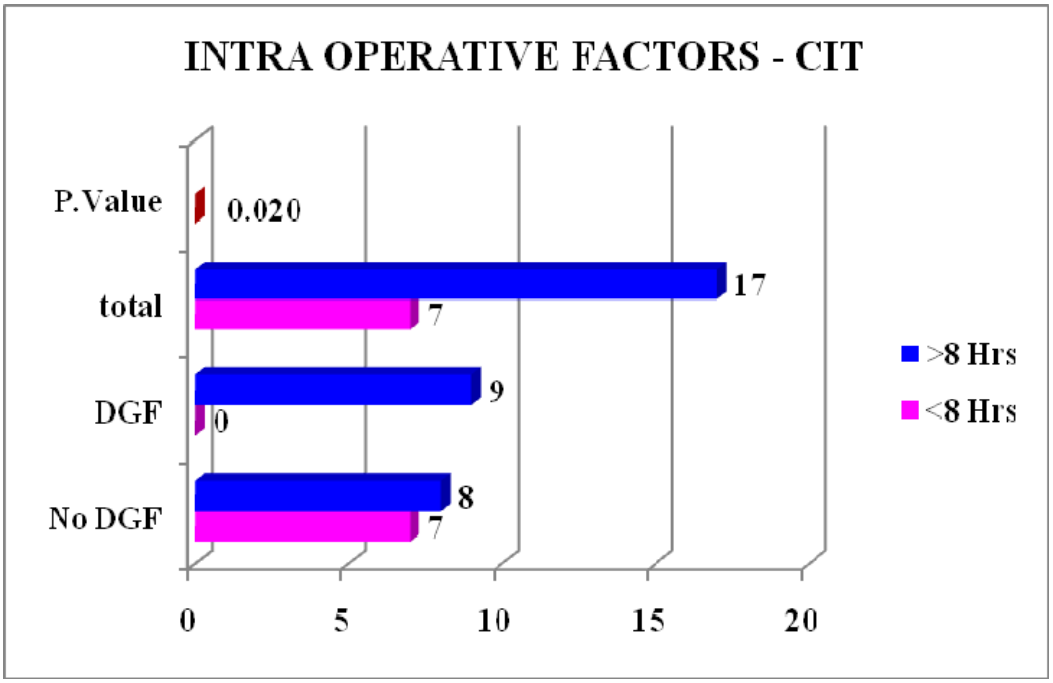
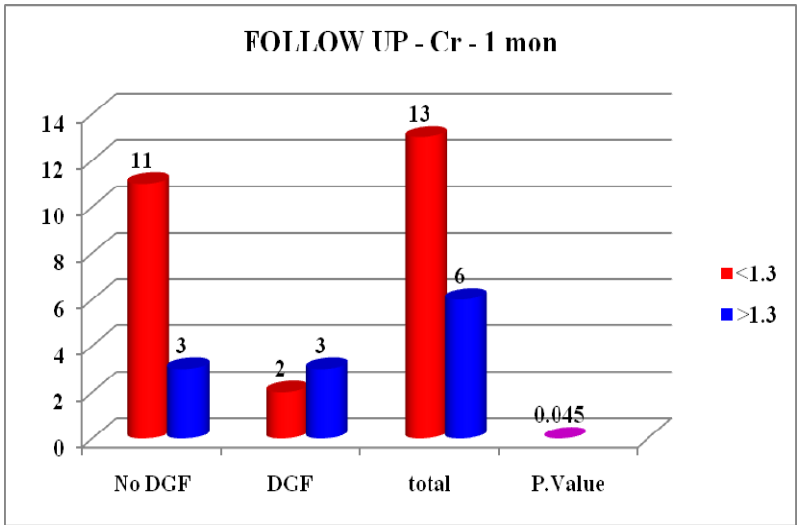


TABLE 10

FOLLOW UP

Variable	No DGF	DGF	total	P Value
Sepsis				
Absent	**14**	**8**	**22**	**0.100**
Present	**2**	**1**	**3**	
Cr-1 mon				
<1.3	**11**	**2**	**13**	**0.045★**
>1.3	**3**	**3**	**6**	
Cr-6 mon				
<1.1	**9**	**1**	**10**	**0.118**
>1.1	**4**	**4**	**8**	


FOLLOW UP - Cr - 1 mon
14
12
10
8
6
4
2
0
11
3
2
3
13
6
0.045
No DGF
DGF
total
P.Value
<1.3
>1.3

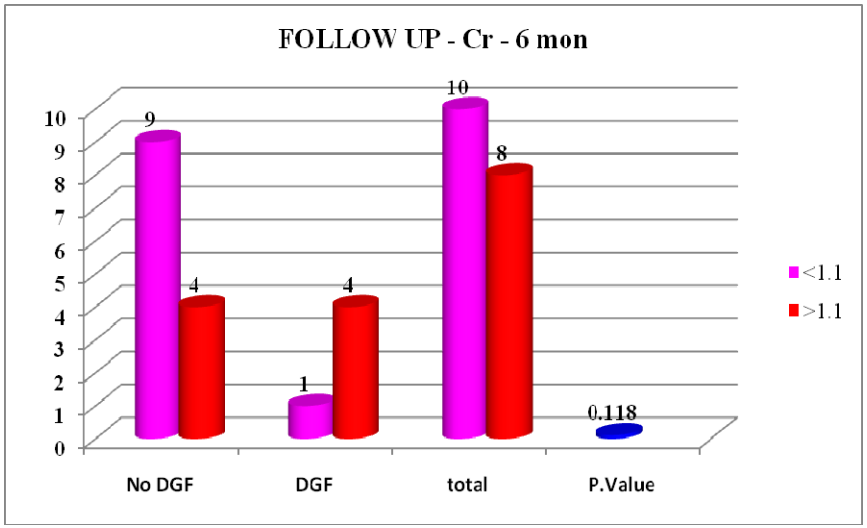

FOLLOW UP - Cr - 6 mon
10
9
8
7
6
5
4
3
2
1
0
9
4
1
4
10
8
0.118
No DGF
DGF
total
P.Value
<1.1
>1.1

TABLE 11

FACTORS INFLUENCING VITAL STATUS OF PATIENTS

Factors	Died	Live	P.value
Recipient Age			
>34	4	10	0.673
<34	4	6	
Gender			
Male	8	14	0.435
Female	8	2	
Dialysis duration			
< 18 mon	4	9	0.556
> 18 mon	4	7	
BMI			
< 21.5	3	10	0.675
>21.5	5	6	
Serology			
Neg	7	15	0.130
HBV/HCV +	1	1	
Sepsis			
Present	13	8	0.100
Absent	1	2	
CIT			
< 8 HRS	2	5	0.076
> HRS	6	11	
DONOR AGE			
< 34	4	10	0.403
> 34	4	6	

CONCLUSION

CONCLUSION

- Cadaver transplantation is the need of the day and is bound to increase with lesser number of live related transplants.
- Optimal HLA mismatching between recipient and donor is not being performed in India.
- Cadaver transplant with increasing experience have more successful outcome.
- Reduction in cold ischemic time by sharing the organ within the City, availability of emergency cross-match facility and performing transplant surgery without delay will improve the graft survival.
- Use of induction therapy might avert early graft failures.
- Patients with CKD Stage-5 on maintenance haemodialysis without voluntary live related donor will benefit immensely by cadaver transplantation.
- In our study, three patients died due to sepsis. Early detection of sepsis, aggressive treatment and possibly regular checking up of CMV status could improve graft outcome in medium term.
- One patient was lost due to surgical cause which would improve with further experience.
- One patient died of early sepsis and fungal infection. Source of infection could have been from cadaver.
- Induction therapy might have averted immediate rejection and graft rupture which occurred in one patient in our study.

- Early detection and aggressive treatment of CMV infection which would avert the graft loss in one of our patients.
- One patient with HCV developed sepsis and died. For him HCV was not treated.
- To conclude, cadaver transplant is a challenge and the results would improve with attacking multiple causes of graft failure.
- Avoiding HCV infection during dialysis, gaining more experience in transplant surgery, surveillance of infection from cadaver donor, induction therapy, regular CMV surveillance, early detection and treatment of sepsis should go a long way in improving the results.
- In addition, the ideal of sharing HLA matched kidney across the State or India remains a distant dream.
- In the absence of such HLA based sharing, more declaration of brain death in our own centre, immediate cross-match facility and early surgery are logistical factors which would improve the deceased donor graft survival.

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kidneys compared with other recipients and wait-listed transplant candidates. *J Am Soc Nephrol* 2001; 12(3): 589 – 597.

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ANNEXURES

- **PROFORMA**
- **MASTER CHART**

RECIPIENT PROFORMA

Name: **Age:** **Sex:** **Blood Group:**

Address: **Occupation:**

Income: **Social Status:** **Wt:** **Ht:** **BMI:**

Medical History: **DM** **Hypertension:**

Lab:

Urea:

Creatinine: **eGFR** **NKD**

Sodium **Potassium** **Bicarbonate** **Chloride**

LFT **Serum Billurubin(T)** **D** **Alb** **Glob** **SGOT** **SGPT** **SAP**

Urine Routine: **24hrs urine Protein** **Urine C/S**

HB **PCV** **Platelets** **TC** **PT** **aPTT**

INR

BT **CT** **FT4** **TSH**

Calcium **Phosphorus** **Uric acid**

Serology: **HBsAg** **Anti Hcv** **HIV** **CMV**

ECHO **Gynecology** **Dermatology**

Dental **ENT** **MGE**

Urology **Endoscopy** **Anesthesia**

Cross matching **USG**

Renal Biopsy **Chest X-ray** **ECG**

On MHD Duration: **Weekly:**

H/o Blood Transfusion

H/o access problem **Doppler iliac vessels:**

Date of Reg: **Date of Transplant:** **Waiting time:**

Intra op events:

Post op events:

Post Transplant Outcome

Immunosuppression

	POD 1	POD 3	POD 5	POD 7	POD 1 MON	POD 3 MON	POD 6 MON
Creatinine							
Urine output							

Discharge Cr:

Post Transplant Ultra sound / Doppler

Tacro level **HB** **PCV**
Platelets

Biopsy **TC**

Urine routine

24 hrs Urine Protein

DONOR PROFORMA

Name: **Age:** **Sex:**

Address: **Occupation:**

Blood Group:

Income: **Social Status:** **Wt:** **Ht:** **BMI:**

Cause of Brain death:

Medical History: **DM** **Hypertension:**

Urea:

Creatinine:

Sodium **Potassium** **Bicarbonate** **Chloride**

LFT **Serum Billurubin(T)** **D** **Alb** **Glob** **SGOT**
SGPT **SAP**

HB **PCV** **Platelets** **PT** **aPTT** **INR**

Serology: **Anti Hcv** **HBsAg** **HIV** **CMV**

Clinical:

BP: **Pulse:** **Ionotropic support:**

Urine Output:

Clamp Time: **CIT:** **Graft abnormality:**
Graft Side:

USG KUB

DOS: Donor Nephrectomy:
Date of Transplant:

Sl.No	Name	Age	Sex	Blood group	Cross Match	Date of Transplant	BMI	Cr- 1wk	Cr-1Mon	Cr- 6Mon	Native Kidney Disease	Hypertension	Diabetes Mellitus	on HD-mon	ECHO
1	**Kondiaraj**	32	M	O+	5.10%	25.10.08	23.1	1.7	1.6	1.4	CGN	YES	NO	36	N
2	**Balaraman**	32	M	B+	5.10%	21.11.08	22.2	1.6	1.2	1.1	CGN	YES	NO	12	LVH
3	**Bala krishnan**	31	M	B+	5.10%	14.1.09	20.2	8.7	1.4	1.3	CGN	YES	NO	6	LVH
4	**Lilly Theresa**	29	M	B+	5.10%	28.1.09	32.2	2	1.3	1.2	CGN	YES	NO	6	N
5	**SasiKumar**	29	M	A+	5.10%	4.2.09	18.7	1	1.2	--	CGN	YES	NO	28	N
6	**Baskar**	38	M	AB+	5.10%	9.3.09	17.9	2.3	1.6	1.7	CGN	YES	NO	3	LVH
7	**Dasan**	48	M	O+	5.10%	14.3.09	25.6	4.2	4	1.6	ADPKD	YES	NO	24	N
8	**Sakthivel**	27	M	B+	5.10%	26.4.09	25.4	13.1	--	--	CGN	YES	NO	30	N
9	**Renuka**	34	M	B+	5.10%	11.5.09	22.2	1.4	1	0.9	IgAN	YES	NO	48	N
10	**Xavier**	43	M	O+	5.10%	21.6.09	25.8	--	--	--	CGN	YES	NO	24	N
11	**GopiKrishnan**	40	M	B+	5.10%	8.8.09	22.1	3.1	1.2	1.3	FSGS	YES	NO	3	EF40%
12	**Subramani**	48	M	O+	5.10%	15.10.09	19.5	0.9	0.9	0.8	CGN	YES	NO	48	N
13	**JayaKumar**	30	M	B-	5.10%	27.10.09	20.8	2.1	1.2	1.1	CGN	YES	NO	1	N
14	**Eswaran**	31	M	B+	5.10%	13.11.09	28.1	7.3	5.6	--	CGN	YES	NO	12	N
15	**Prema**	35	F	A-	5.10%	4.12.09	31.2	7.4	1	1.2	CGN	YES	NO	24	LVH
16	**Revathy**	24	F	O+	5.10%	12.12.09	17.6	1.2	1.3	1.1	CGN	YES	NO	12	LVH
17	**Devaraj**	46	M	O+	5.10%	16.12.09	18.1	1.3	1.2	1.1	CGN	YES	NO	12	LVH
18	**Palani**	37	M	O+	5.10%	27.12.09	19.3	1.6	1.4	1.2	CGN	YES	NO	24	LVH
19	**Elawarasan**	22	M	AB+	5.10%	20.2.10	17.4	1	0.9	--	CGN	YES	NO	24	N
20	**Riyaz ali**	25	M	B+	5.10%	27.2.10	18.9	0.9	0.8	0.8	CGN	YES	NO	12	N
21	**Dass Prakash**	31	M	B+	5.10%	19.3.10	26.6	1.6	1.2	1	CGN	YES	NO	6	N
22	**Rajan**	36	M	B+	5.10%	6.4.10	22.3	5.3	--	--	CGN	YES	NO	3	N
23	**Devi**	29	F	O+	5.10%	11.4.10	17.3	1.2	0.9	0.9	CGN	YES	NO	1	LVH
24	**Nirmala**	34	F	AB+	5.10%	14.4.10	18.7	6.5	0.9	0.8	CGN	YES	NO	36	LVH

Sl.No	INTRA OP	POST OP	Tacro Level	Serology	Doppler	DONOR	Age	Sex	Blood group	Diabetes Mellitus	Hypertension	Graft side	CIT
1	NIL	NIL	10	NEG	N	**Radhakrishnan**	49	M	O+	NO	NO	LEFT	10
2	NIL	NIL	8	NEG	N	**Gnanaprakasem**	26	M	B+	NO	NO	LEFT	8
3	NIL	HD-3	10	NEG	N	**Premkumar**	48	M	B+	NO	NO	LEFT	12
4	Bleeding	NIL	3	NEG	N	**Jeevarathinam,**	56	F	B+	NO	NO	LEFT	12
5	NIL	NIL	16.3	NEG	N	**Suganya**	15	F	A+	NO	NO	LEFT	3
6	NIL	ACC.HT	10	NEG	N	**Asha**	20	F	AB+	NO	NO	LEFT	10
7	NIL	SEPSIS	2.9	HBV+	N	**Jeyanthi Reddy**	39	M	O+	NO	NO	LEFT	10
8	Mottling	Nephrectomy	3.1	HCV+	N	**Chandru**	27	M	B+	NO	NO	LEFT	10
9	Venous leak	NIL	14.2	NEG	N	**Dharani**	19	F	B+	NO	NO	LEFT	7
10	EIA	Rt leg ischemia		NEG	N	**Sivaprakasam**	42	M	O+	NO	NO	LEFT	12
11	Hilum anas	Hypotension	18	NEG	N	**John rayan**	57	m	B+	NO	NO	LEFT	10
12	NIL	NIL	8	NEG	N	**Vinoth Kumar**	28	M	O+	NO	NO	LEFT	11
13	NIL	Pancreatitis	11	NEG	N	**Iyyappan**	28	M	B+	NO	NO	RIGHT	10
14	Hypotension	persistent DT	13	NEG	N	**Loganathan**	23	M	B+	NO	NO	RIGHT	12
15	Hypotension	Ionotropes	3.8	NEG	N	**JaiAnand**	18	F	A+	NO	NO	LEFT	12
16	NIL	SEPSIS/ARDS	15	NEG	N	**Palanivel**	24	M	O+	NO	NO	RIGHT	11
17	NIL	NIL	10.3	NEG	N	**chandran**	56	M	O+	NO	NO	LEFT	11
18	NIL	NIL	15.2	NEG	N	**Jayabharthi**	15	F	O+	NO	NO	LEFT	3
19	NIL	NIL	9	NEG	N	**Vijay**	12	M	AB+	NO	NO	LEFT	5
20	NIL	NIL	12	NEG	N	**Venkatasen**	29	M	O+	NO	NO	RIGHT	5.5
21	NIL	NIL	10.9	NEG	N	**Kuppan**	45	M	B+	NO	NO	RIGHT	10
22	NIL	Fungal sinusitis		NEG	N	**Malliga**	34	F	B+	NO	NO	RIGHT	8
23	NIL	Stitch abscess	9.3	NEG	N	**Lakshmi**	45	F	O+	NO	NO	RIGHT	9
24	NIL	ATN	9.8	NEG	N	**Rajadurai**	19	M	A+	NO	NO	LEFT	8

Sl.No	Graft anamoly	Cause of Death	Graft function	Patient Status	Graft Biospy	Perfusion Sol	Immuno suppression	Tx - I/II
1	3 RA/CUFF	RTA	N	DIED(18/1/10)	CAN	HTK	T/M/P	I
2	NIL	RTA	N	DIED(20/1/10)	NIL	HTK	T/M/P	I
3	3RA	RTA	DGF	ALIVE	NIL	HTK	T/M/P	I
4	NIL	RTA	N	ALIVE	NIL	HTK	T/M/P	I
5	2RA	RTA	N	DIED(9/4/9)	NIL	HTK	T/M/P	I
6	NIL	RTA	N	DIED(15/6/9)	HUS	HTK	T/M/P	I
7	NIL	RTA	DGF	DIED(30/4/9)	NIL	HTK	T/M/P	I
8	NIL	RTA	DGF	ALIVE	NIL	HTK	T/M/P	I
9	NIL	RTA	N	ALIVE	NIL	HTK	T/M/P	I
10	2 RA	RTA	DGF	DIED(22/6/9)	NIL	HTK	T/M/P	I
11	NIL	RTA	DGF	ALIVE	NIL	HTK	T/M/P	I
12	NIL	RTA	N	ALIVE	NIL	HTK	T/M/P	I
13	NIL	RTA	N	ALIVE	NIL	HTK	T/M/P	I
14	2 RV	Fall from ht	DGF	DIED (7/6/10)	ACR/ AHR	HTK	T/M/P	II
15	2 RA	Fall from ht	DGF	ALIVE	NIL	HTK	T/M/P	I
16	NIL	RTA	N	ALIVE	NIL	HTK	T/M/P	I
17	NIL	RTA	N	ALIVE	NIL	HTK	T/M/P	I
18	NIL	RTA	N	ALIVE	NIL	HTK	T/M/P	I
19	NIL	RTA	N	ALIVE	NIL	HTK	T/M/P	I

Cr.1 wk : **Creatinine value at 1 week Post Operation**

Cr.1 mon : **Creatinine value at 1 month Post Operation**

Cr.6 mon : **Creatinine value at 6 months Post Operation**

Intra OP : **Intra Operative events**

Post OP : **Post operative events**

Tacro level : **Tacrolimus through level**

CIT : **Cold Ischemic Time**

Tx - I/II : **First or Second Transplant**

CGN : **Chronic Glomeulo Nephritis**

ADPKD : **Autonomal Dominant Polycyotic Kidney disease**

IgAN : **IgA Nephropathy**

LVH : **Left Ventricular Hypertrophy**

RTA : **Road Traffic Accident**

T/M/P : **Tacrolimus, Mycophenolate mofetil and Prednisolone**

20	2 RA	RTA	N	ALIVE	NIL	HTK	T/M/P	I
21	NIL	RTA	N	ALIVE	NIL	HTK	T/M/P	I
22	NIL	RTA	DGF	DIED(16/4/10)	NIL	HTK	T/M/P	I
23	NIL	RTA	N	ALIVE	NIL	HTK	T/M/P	I
24	NIL	Fall from ht	DGF	ALIVE	NIL	HTK	T/M/P	I